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BY

GEORGE E. DE SCHWEINITZ, M.D., LL.D. (Univ. of Pa.)

Professor of Ophthalmology in the University of Pennsylvania; Ophthalmic Surgeon to the University Hospital; Consulting Ophthalmic Surgeon to the Philadelphia Polyclinic Hospital, the Philadelphia General Hospital, and the Orthopedic Hospital and Infirmary for Nervous Diseases

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PREFACE TO THE EIGHTH EDITION

In the Eighth Edition of this text-book its chapters have been revised thoroughly in an endeavor to include due reference to the important ophthalmic observations and therapeutic measures which have been made and recommended during the last three years.

A new feature in this edition is the introduction of the metric equivalents of the doses of the remedies and the strengths of the solutions previously recorded only according to the old system. Reference to the following subjects appears for the first time: Clifford Walker's Method of Testing the Visual Field; Squirrel Plague Conjunctivitis; Swimming Bath Conjunctivitis; Anaphylactic Keratitis; Family Cerebral Degeneration with Macular Changes; the Ocular Symptoms of Disease of the Pituitary Body; Sclerectomy with a Punch (Holth's Operation); Preliminary Capsulotomy (Homer Smith's Operation); Iridotasis (Borthen's Method); Thread Drainage of the Anterior Chamber (Zorab's Operation); Extraction of Cataract in the Capsule after Subluxation of the Lens with Capsule Forceps (Stanculeanu's Method, Arnold Knapp's Method); Capsulomuscular Advancement with Partial Resection (Ziegler's Method); Tenotomy of the Inferior Oblique; Window Resection of the Nasal Duct (West's Operation).

A portion of the chapter on Iritis has been rewritten, especially as it relates to Autotoxemic Iritis and Iritis Secondary to Mucous Membrane Infections. Lieut.-Col. Elliot has very kindly written the description of Corneoscleral Trephining, ordinarily known as Elliot's Operation. To Dr. William M. Sweet the author is indebted for a description of his revised method of localizing foreign bodies within the eye by means of the *x*-rays. As in previous editions, the section devoted to Skiascopy, or the Shadow Test, is from the pen of Dr. Edward Jackson. A number of new illustrations have been inserted. The entire work has been reset and appears in a new and greatly improved form.

The author trusts that these additions and alterations will prove to be satisfactory, and he desires again to express his high appreciation of the cordial reception which has thus far been accorded to his work.

G. E. DE S.

1705 Walnut Street,
Philadelphia, Pa.
May, 1916.

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DISEASES OF THE EYE

CHAPTER I

GENERAL OPTICAL PRINCIPLES

Transmission of Light.—By light is meant that physical force or form of energy which, acting on the sentient elements of the retina, causes the mental perception of the specific energy, that is, *sight* or *vision*.

From each point of the surface of a luminous body *light* or *rays of light* proceed in straight lines in all directions, and in order to explain the transmission of light it is assumed that throughout the universe there exists an exceedingly tenuous matter to which the term *ether* is applied (see page 20). Exactly what the vibrating disturbances are which constitute light is not certainly known.

Refraction.—By refraction of light is meant the alteration which takes place in the direction of luminous rays, which pass obliquely from one medium into another of different density.

A ray of light passing through air keeps the same direction until it strikes obliquely the surface of a denser medium, when its course is changed toward the perpendicular to that surface. If this denser medium is a piece of glass bounded by parallel sides, the ray, as it passes through the second surface, is bent back again into the rarer medium.

Rays passing from a denser into a rarer medium are deviated from the perpendicular. The ray now has a direction parallel to its original course; the sides being parallel, the deviation at each surface is equal in extent, but opposite in direction (Fig. 1).

If the denser medium is bounded by oblique surfaces, the deviation at the second surface does not restore the ray to its original direction, but it still more increases the alteration of its direction (Fig. 2).

Index of Refraction.—The deviation of the ray from its course depends upon the difference in the density of the two media.

A ray passing obliquely from one medium into another of the *same* density is not bent from its course. The relative resistance of a substance to the passage of light is expressed by its *index of refraction*. The absolute index of refraction is its resistance as compared with vacuum; but as there is very little difference between the indices of refraction of air and of vacuum, air is considered as 1 for all calculations in lenses.

As the difference in the density of the two media increases, the ray is bent more sharply from its course, and the angle it forms with the perpendicular after refraction by a denser medium is proportionably smaller than the angle formed by the ray before refraction.

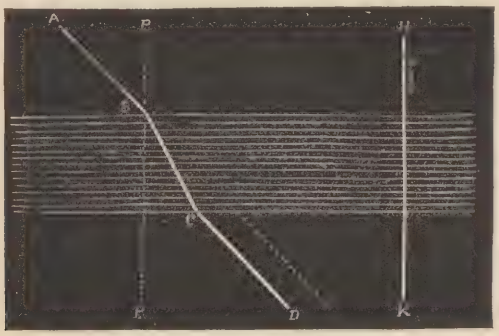


Fig. 1.—Refraction of light through a plate of glass bounded by plane surfaces which are parallel: *A–B* is the incident ray; *B–C*, the same ray, refracted by the first surface, nearer to the perpendicular, *P–P*; *C–D*, the same ray, refracted by the second surface, becomes parallel to *A–B*, its original direction. The ray *H–K*, perpendicular to the surfaces *B* and *C*, undergoes no refraction.

The angle formed by the ray with the perpendicular to the surface of the second medium is called the *angle of incidence*—angle *I*. The angle formed by the ray with the perpendicular after refraction is called the *angle of refraction*—angle *R*. The sine of the angle of incidence, divided by the sine of the angle of refraction, gives the index of refraction. Glass used in the manufacture of spectacles has an index of refraction of about 1.53.

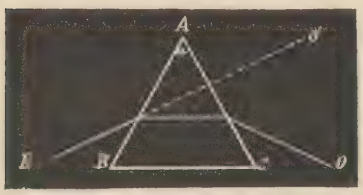


Fig. 2.—Refraction through a denser medium having oblique surfaces. At each surface the ray is bent toward the base of the figure.

Prisms.—A *prism* is a portion of glass or other refracting substance bounded by two plane surfaces which are inclined to each other, forming an angle, which is called the *refracting angle*, or simply the *angle* of the prism (Fig. 2, *a*), and is expressed in degrees. Prisms are often designated by the numbers of degrees in the refracting angle.

The sides of the prism converge to a thin edge at one extremity, called the *apex* (Fig. 2, *A*); at the other extremity they diverge from each other and form the *base* (Fig. 2, *B–C*).

Refraction Through a Prism.—If a ray of light from an object (Fig. 2, *O*) passes through a prism the refractive index of which is greater than air, the deviation is always from the apex toward the base of the prism.

To the eye of an observer placed at the other side of the prism (Fig. 2, *E*) the refracted ray seems to come from the direction of the

apex (Fig. 2, *O'*), since a ray is projected backward over the course given to it by its last refraction, and a single object appears double if, with both eyes open, a prism of sufficient strength is placed before one of them. The angle which the ray in this last direction forms with the ray in its original direction is called the *angle of deviation.*

When one eye, on account of muscular weakness, is unable to direct its visual line to the point of fixation, a prism will alter the direction of the ray from the point of fixation so that it coincides with the visual line of the weaker eye. The refractive properties of a prism are utilized to test the strength of the ocular muscles (see pages 74 and 555), to neutralize the diplopia caused by abnormal deviation of the visual line—for example, in paralytic strabismus and in the treatment of heterophoria (see page 559)—to detect malingerers who feign monocular blindness (see page 505), and in the application of the Wildbrand-Saenger test to determine the situation of the lesion in hemianopsia (see page 518).

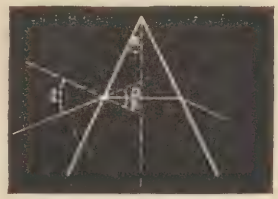


Fig. 3.—Deviation produced by a prism: *I*, Angle of incidence; *R*, angle of refraction; *D*, angle of deviation; $R + D = I$; *D* equals in weak prisms about $\frac{1}{2}$ of *R* (Jackson).

Angle of Deviation.—The angle of deviation is the angle formed by the incident ray with the refracted ray. The amount of this angle is somewhat more than one-half of the refracting angle of the prism for all prisms between 1° and 10°, but for practical purposes the two may be considered equal. Above this the deviation rapidly increases.

When the angle of incidence, formed by a ray in the interior of a prism, amounts to 40° 49′, the angle of refraction equals 90°; the angle of deviation, the difference between the two, then equals 49° 11′. When the refraction which takes place at each surface of a prism is equal, the minimum amount of deviation is present. When the ray is perpendicular to one surface, the angle of incidence at the second surface equals the angle of the prism; the deviation is greater in this case, as all the refraction takes place at one surface. A table of the minimum deviation of prisms is given on page 20.

Numbering of Prisms.—The designation of prisms by their angular deviation, instead of by their refracting angles, was urged by Dr. Edward Jackson, of Denver, before the Ninth International Medical Congress. Two methods of accomplishing this have been proposed:

Dennett's Method: The Centrad.—Dr. William S. Dennett's calculation has for its base an arc called the *radian*, whose length equals the radius of its curvature. Such an arc equals 57.295°. A prism which will produce an angular deviation of the one-hundredth part of this arc is called *one centrad.* The deviation of such a prism would, therefore, be 0.57295°. The merit of this method consists in the uniformity of the deviation, 10 centrads having exactly ten times the deviation of 1 centrad. The deviations are so many hundredths of the radius measured on the arc.

Prentice's Method: The Prism-diopter.—Mr. Charles F. Prentice proposes, as the standard of deviation, a prism which shall deflect a ray of light 1 cm. at a plane 1 meter distant—that is, the hundredth part of the radius measured on the tangent. This he calls the *prism-diopter*. The value of the centrad and prism-diopter are given below (see table).

There are two practical advantages connected with the method of Mr. Prentice which also can be applied to the centrad. The prismatic deviation of a decentered lens may be very readily found, as Prentice has shown by the following rule: If a lens be decentered 1 cm., the prismatic deviation of the lens will be equal to as many prism-diopters as the number of diopters in the lens. Thus, if a 4-diopter lens be decentered 1 cm., the prismatic deviation will be 4 prism-diopters, or 4 centrads, since centrad and prism-diopter exactly equal each other. The same lens decentered 0.5 cm. would produce 2 prism-diopters or centrads of deviation.

Table of Relative Values of Centrads and Prism-diopters, Prepared by James Wallace.

Centrads.	Prism-diopters.	Refracting angle of prism required.
1	1	1.06°
2	2.0001	2.16°
3	3.0013	3.24°
4	4.0028	4.32°
5	5.0045	5.40°
6	6.0063	6.47°
7	7.0115	7.54°
8	8.0172	8.62°
9	9.0244	9.68°
10	10.0333	10.73°
15	15.114	16.1°
20	20.270	21.13°
40	42.288	39.0073°

The prisms represent the minimum deviation with an index of refraction of 1.53.

The relation to the meter angle (see page 44) is also very simple. One-half the interpupillary distance is the sine of the meter angle. The ratio of this to the point of fixation in hundredths gives nearly the number of prism-diopters, or centrads of deviation, embraced in any number of meter angles. For example, if the interpupillary distance is 60 mm., one-half of this is 30 mm.; assuming the amount of convergence to be 4 meter angles, 25 cm., or 250 mm., is the distance of the point of fixation. The deviation of the visual line then is 30 in 250, or 12 in 100 = 12 centrads, or 12 P. D. For small arcs the tangent and the sine agree very closely with the arc. Four meter angles of convergence then represent 12 centrads of deviation or 12 prism-diopters.

Rays of Light.—Any luminous point diffuses light in all directions in straight lines called *rays*. As the rays proceed from the luminous source, those which diverge from one another become more widely separated (Fig. 4).

If a circular aperture 1 cm. in diameter be made in a metal plate and a luminous point be placed at different distances from it—for example, at 1 meter and at 10 meters—the rays coming from 10 meters, which pass through the aperture, will be less diverging than those which come from 1 meter. A cone of light will pass through the aperture in each case, but the shape of it will be different according to the distance of the light from the aperture in the screen. When the round hole, 1 cm. in diameter, is 1 meter distant from the point of light, the cone has a base 1 cm. in diameter, and the apex is situated in the luminous point 100 cm. distant. The rays have diverged 1 cm. in traveling 100; the metal plate has cut off all other rays having a greater divergence. If the cone of light passes through the aperture and falls upon a distant wall, the cone will preserve the same proportions—viz., the base will be $\frac{1}{100}$ of the altitude. If the wall be 5 times the distance of the screen from the light, a luminous circle 5 cm. in diameter will be formed upon the wall. If, now, the light is removed to a point 10 meters from the screen (1000 cm.), a cone of light is formed whose base is 1 cm. and whose altitude is 1000. The rays which pass through the aperture have now only $\frac{1}{10}$ of the divergence of the rays in the former case; the base of the cone is $\frac{1}{1000}$ of the altitude. The cone of light will now form a circle on the wall 5 meters beyond the aperture, only 1.5 cm. in diameter. If the point of light be at a very great distance, there will be no

Fig. 4.—Divergence of rays from a luminous source (Loring).

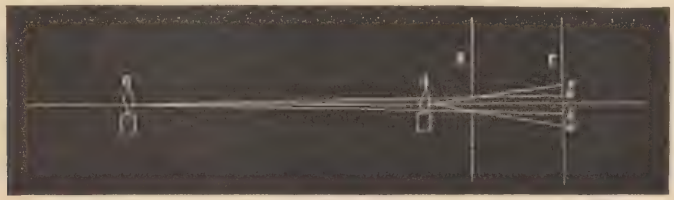


Fig. 5.—Rays diverging from the candle A pass through the aperture in the screen S, and form the cone of light whose base is the distance a–a'. Rays from a more distant candle, B, having a greater divergence than b–b', are intercepted by the screen S (Wallace).

difference in the size of the luminous circle and the aperture in the screen; the size of the circle remains about 1 cm. on the wall at 5 meters from the screen. The rays, therefore, have a nearly parallel direction. This is shown in Fig. 5.

Rays which enter the pupil of the eye from a point 6 meters distant have so little divergence that they may be considered parallel. The average size of the pupil being 4 mm., the divergence is only $\frac{4}{6000}$. All rays diverging more widely than this are excluded by this width of the pupil.

The relation to the eye of rays diverging from 6 meters or coming from an infinite distance is practically identical, but for lenses of long focal distance and large aperture an infinite distance is required in order to obtain parallel rays. Thus the sun and stars are so remote that the rays coming from them have no appreciable divergence, and they are considered parallel.

Parallel rays must emanate, as has been explained before, from a distant object. They are brought together by a lens at its principal focus. Conversely, rays which diverge from the principal focus of a lens are parallel to one another after being refracted by the lens

Divergent rays emanate from an object nearer than infinity. A greater refractive power must be exercised to bring them together at the same distance behind a lens than is required for rays which are parallel; consequently, divergent rays are united at a point farther than the principal focus. The nearer the point of divergence lies to the lens, the farther away from the lens is the point where the rays converge to a focus.

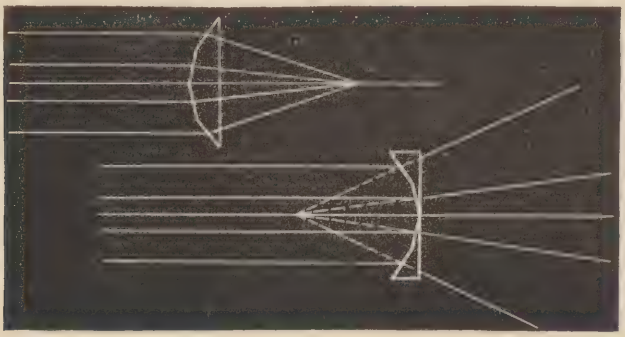


Fig. 6.—Lenses as prisms.

Convergent rays do not exist in nature. Only such rays are convergent which have passed through a convex lens or have been reflected from a concave mirror.

Significance of the Different Rays.—The refraction of the eye is determined by the character which the rays must have in order to be brought to a focus on the retina.

An *emmetropic eye*, with relaxed accommodation, requires rays to be parallel in order that they shall meet on the retina.

A *myopic eye* requires rays to diverge from some near point in order to meet on its retina.

A *hyperopic eye* requires rays which already have convergence to some point in order to unite them on its retina.

An emmetropic eye emits parallel rays.

A myopic eye emits convergent rays.

A hyperopic eye emits divergent rays.

Lenses.—A lens is a portion of glass or other transparent substance bounded by two curved surfaces, or by one curved surface and one plane surface. The curved surfaces are convex, elevated in the center, and thin at the edge; or they are concave, hollowed out in the center and thick at the edge.

A lens may be regarded as a series of prisms with the refracting angles increasing in value from the center toward the periphery.

In a *convex lens* the bases of the prisms are directed toward the center of the lens, and rays, therefore, are refracted toward the axis which passes through the center. In a *concave lens* the bases of the prisms are directed away from the center, and rays, therefore, are refracted away from the axis. As the angles increase from the center outward, the peripheral rays will be refracted more than the central rays. The result of this is that in a convex lens the rays after refraction converge to the same point, the increased bending of the more peripheral rays just sufficing to compensate for their greater distance from the axis. In a concave lens the rays diverge more widely as they

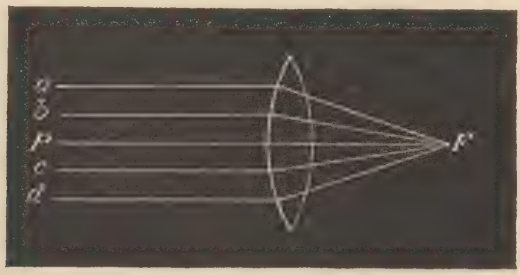


Fig. 7.—Principal focus of a convex lens. The parallel rays *a*, *b*, *c*, *d* are refracted by the lens so as to unite at the point *F* on the axis *P*. The ray *P* undergoes no refraction. *F* is the principal focus.

pass through the peripheral parts of the lens, with the result of making them appear to have diverged from a common point.

Focus of a Convex Lens.—The point to which rays converge after refraction by a convex lens is called its *focus*.

Principal Focus of a Convex Lens.—The principal focus of a lens is the focus for parallel rays. As the most distant rays are only parallel, never convergent, the principal focus is the shortest focus, unless the lens is combined with another convex lens or concave mirror. Rays diverging from the principal focus of a lens are rendered parallel after passing through the lens, and come to a focus at an infinite distance.

Conjugate Focus of a Convex Lens.—When rays diverge from any point nearer than infinity, they are brought together at a point on the other side of the lens farther than the principal focus. The point from which rays diverge and the point to which they converge are called *conjugate foci*. As the point of divergence approaches the lens the point of convergence recedes; when the point of divergence is

at twice the focal distance of the lens, the point of convergence is at an equal distance on the other side. The *conjugate foci* are now equal.

As the point of divergence approaches still closer the point of convergence is at a greater distance, until, when the point from which the rays diverge is at the principal focus, the rays converge at an infinite distance.

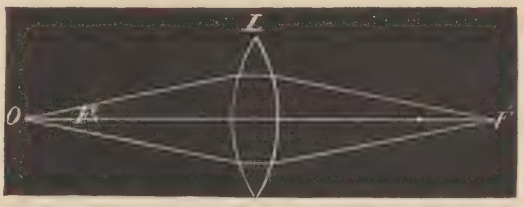


Fig. 8.—Conjugate focus of a convex lens. The two dots in the axis represent the principal foci, one being marked *F*. Rays diverging from *O* converge after refraction to the point *F'*, farther than the principal focus. Rays from *F'* also converge after refraction to *O*. *O* and *F* are conjugate foci.

Rays diverging from either of these points converge toward the other. When rays diverge from a point whose distance is equal to or greater than the principal focus, the conjugate focus is *positive*. When the distance is less than the principal focus, the conjugate focus is *negative*.

Virtual Focus of a Convex Lens.—When rays diverge from some point nearer to a lens than its principal focus, the rays after refraction still continue divergent. These divergent rays, if traced backward,

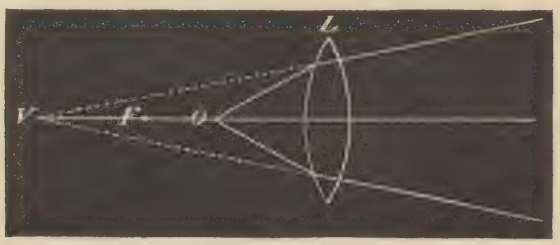


Fig. 9.—Virtual focus of a convex lens. Rays from the point *O*, less than the principal focal distance, diverge after refraction as if they came from the point *V*. *V* is the virtual focus of *O*.

would meet in a point on the same side of the lens from which they diverged. This point is called a *negative*, or *virtual*, focus, because the rays do not really meet here, but are given a direction by the lens as if they had diverged from this point (Fig. 9). Therefore the point from which rays diverge and the point to which they converge are focal points.

Foci of Concave Lenses.—The foci of concave lenses for parallel or divergent rays are virtual, or negative. They are the points from which the rays seem to diverge after passing through the lens.

Principal Focus of a Concave Lens.—When parallel rays fall upon a concave lens they are rendered divergent. If these rays be traced backward, they will seem to have diverged from a point near the lens. This point is the *principal focus* (Fig. 10).

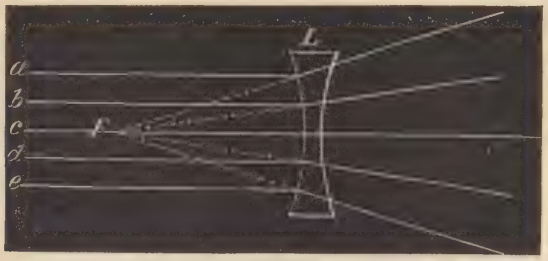


Fig. 10.—Principal focus of a concave lens. Parallel rays *a*, *b*, *d*, *e*, after refraction by the concave lens *L*, are rendered divergent as if they came from the point *F* on the axis *c*. The ray *c* is not refracted. *F*, the principal focus of a concave lens, is virtual.

Conjugate foci of concave lenses are also virtual and found in a similar manner.

Formation of Images by a Lens: Optical Center.—In the lens (Fig. 11) the point *O* on the axis is called the *optical center*. Any ray

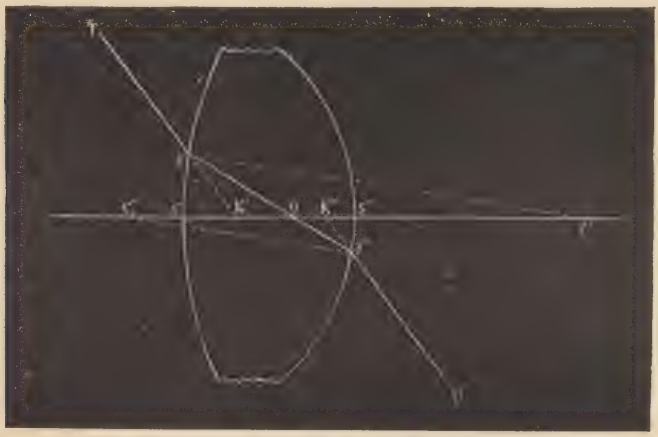


Fig. 11.—*O*, Optical center of lens. The point *C″* is the center of curvature for the surface *S″*. The point *C′* is the center of curvature for the surface *S′*. A ray passing from *C″* to *C′* would be perpendicular to both surfaces. It would pass through without deviation. This ray is called the *axial ray*, or *axis*.

The radii *C″–J″* and *C′–J′*, being parallel, a ray in the lens passing in the direction *J′–J″* must form equal angles at the two surfaces. The point where this ray intersects the axis is the *optical center* (Landolt).

passing through this point is refracted equally at both surfaces, since it forms equal angles with the radii of the two surfaces. The direction of the ray is, therefore, the same after refraction by the second surface as it was before refraction by the first. For thin lenses it may be said

that any ray directed to the optical center passes through without deviation. These rays are called *secondary axes.*

The ray drawn from any point in an object to the optical center of a lens gives the line on which the image of the point is to be found. A ray from the same point in the object, passing parallel to the axis of the

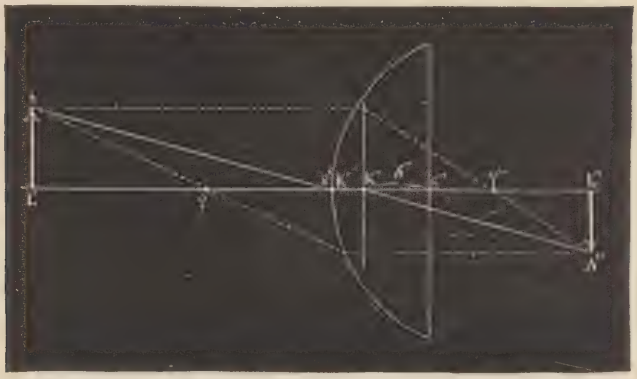


Fig. 12.—Position and size of image formed by convex lens. The ray A, K', from the point A, being directed to the optical center of the lens, continues its course in a parallel direction, K''–A''. Another ray passing from A parallel to the axis L', L'', is refracted through φ'', the principal focus, and, intersecting the ray A–K''–A'', determines the position of the image of the point A. Still another ray passing from A through the anterior principal focus φ', after refraction, is parallel with the axis L'–L'', and meets the other rays in the point A'' (Landolt).

lens, would be refracted through the principal focus of the lens, since the principal focus is the focus for parallel rays (Fig. 12).

In order to find the position and size of an image formed by a lens it is only necessary to draw two lines from each extremity of the object: one passes through the optical center of the lens, and the other, parallel

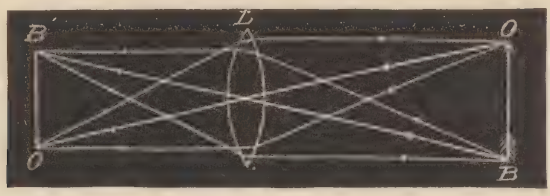


Fig. 13.—Image formed by a convex lens: O–B is the object; O'–B' is the inverted image.

with the axis of the lens, would be refracted to the principal focus. The *position of the image* is found at the points where these lines intersect.

The size of the image is proportional to the size of the object as the distance of the image from the optical center is to the distance of the object from the optical center. When the object is situated at a greater distance from the lens than its principal focus, the image is a real, inverted one.

In the figure (Fig. 13) *O–B* is the object; the rays diverging from *O* intersect in *O′*, which is the position of the image of the point *O*. Similarly the rays from *A* unite in *B′*, the position of the image of the point *B; B′–O′* is the image of *O–B*.

When the object is situated nearer to the lens than its principal focus, the image is a virtual, erect one.

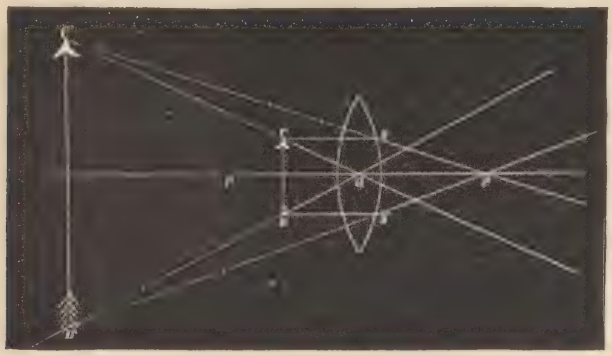


Fig. 14.—Virtual image of a convex lens: *C–D* is the object; *C′–D′* is the virtual image, erect and magnified.

The *virtual* image of a *convex* lens appears to be at the point from which the rays refracted by the lens seem to have diverged (Fig. 14). From the point *C*, of the object *C–D*, the ray *C–S* is parallel to the axis. It, therefore, is refracted to the principal focus, *P*. The ray *C–O* passes through unchanged. By projecting these rays backward they meet in *C′*, the image of the point *C*. The rays from the point *D* seem to have diverged from *D′*. An enlarged, *erect* image is thus formed in *C′–D′*.

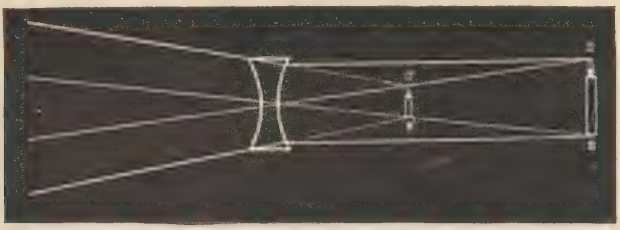


Fig. 15.—Virtual image of a concave lens: *O′–B′* is the virtual image of the candle, *O–B*, erect and diminished in size.

The image formed by a *concave* lens is mostly *virtual* and diminished. Two rays, proceeding from a point *O*, in the object, one parallel to the axis, which seems, after refraction, to have diverged from the principal focus, and is traced backward, and the other, which is directed to the optical center, at their intersection, denote the position of this point in the image (Fig. 15). The enlarged image formed by a convex lens, and the diminished image formed by a concave lens, as described

in the preceding paragraph, are among the most obvious effects of such lenses as they are ordinarily used. It must be remembered, however, that convex lenses are not essentially magnifiers nor concave lenses essentially minifiers, inasmuch as their effect on images depends upon their position with reference to optical systems which they supplement.

Focal Distance of a Lens.—The distance from the optical center of a lens to the focal point is called the *focal distance.*

The length of this depends upon the radii of curvature of the surfaces of the lens and on its index of refraction. Representing the radius by r, the index of refraction of the lens by n, that of air being 1, $F = \frac{r}{2\,(n-1)}$ is the formula for obtaining the focus of a bispheric convex or concave lens. The formula for a planospheric lens is $F = \frac{r}{n-1}$. The refraction is effected at one surface if the rays are parallel as they enter or pass from the plane surface; otherwise refraction occurs at the plane as well as at the curved surface.

Numeration of Lenses.—The refractive power of a lens is the inverse of its focal distance. If the refractive power of a lens whose focal distance is 1 meter is represented by 1, then a lens whose focal distance is 2 meters has only one-half the refractive power of the first, since the rays are not bent so sharply by the second lens. Again, if a lens bends rays so sharply that they meet the axis at 0.5 meter distance, its refractive power is twice that of a lens of 1 meter focus.

The focus of a biconvex lens (with equal radii), made of glass with an index of 1.50, has the same length as the radius of curvature.

$$F = \frac{r}{2\,(n-1)} = \frac{r}{2\,(1.50-1)}$$
$$F = r.$$

Glass used in spectacle lenses has an index of 1.53, consequently—

$$F = \frac{r}{1.06}$$
$$r = 1.06\,F.$$

In the old system the lenses were marked according to their radii of curvature in Paris inches, and the focal distance was somewhat less than the radius of curvature. As all the lenses in use had longer focal distances than 1 inch, they were fractions of the refractive power of a lens of 1 inch focus—viz., $\frac{1}{2}$, $\frac{1}{4}$, $\frac{1}{8}$, $\frac{1}{16}$, etc.

In 1867 Nagel proposed to number lenses by their refractive power. By adopting as a standard a lens of longer focal distance than 1 inch—viz., 1 meter (40 inches)—the greater number of lenses are made multiples of refractive power of the standard, and are based on their focal lengths in meters and fractions of a meter, instead of being based on their radii of curvature.

The term *diopter* was proposed by Monoyer for a lens of 1 meter

focus. A lens of 2 meters focus is only $\frac{1}{2}$ the refractive power, or 0.50 D. The present scale of lenses comprises a series from 0.12 to 22 D. Between 0.12 and 1.25 D the lenses have an interval of 0.12 D. From 1.25 to 5 D the interval is 0.25 D; from 5 to 8 D an interval of 0.50 D; from 8 to 18 D an interval of 1 D; and from 18 to 22 D the interval is 2 D. This uniformity in the intervals between the lenses is an important advantage over the old system, in which the lack of uniformity in this respect was a conspicuous feature.

To find the focal length of any lens in the dioptric system divide 1 meter, or 100 cm., by the number of diopters: thus the focal length of a lens of 5 D is $\frac{100}{5} = 20$ cm.

	Number of lens in diopters.	Focal distance in millimeters.	Focal distance in English inches.	Nearest corresponding lens in old system.
	0.12	8000	314.96	
	0.25	4000	157.48	144
	0.37	2666	104.99	
	0.50	2000	78.74	72
Interval of 0.12 D.	0.62	1600	62.99	60
	0.75	1333	52.5	48
	0.87	1143	44.99	42
	1	1000	39.37	36
	1.12	888	34.99	
	1.25	800	31.5	30
	1.5	666	26.22	24
	1.75	571	22.48	
	2	500	19.69	20
	2.25	444	17.48	18
	2.50	400	15.75	16
	2.75	363	14.31	15 or 14
	3	333	13.12	13
Interval of 0.25 D.	3.25	308	12.11	12
	3.50	285	11.25	11
	3.75	267	10.49	10
	4	250	9.84	9
	4.25	235	9.26	8
	4.50	222	8.74	8
	4.75	210	8.29	
	5	200	7.87	
	5.50	182	7.16	7
	6	166	6.54	
Interval of 0.5 D.	6.50	154	6.06	6
	7	143	5.63	5
	7.50	133	5.25	
	8	125	4.92	
	9	111	4.37	4.5
	10	100	3.94	4
	11	91	3.58	3.5
	12	83	3.27	3.25
Interval of 1 D.	13	77	3.03	3
	14	71	2.8	2.75
	15	66	2.64	
	16	62	2.44	2.5
	17	59	2.32	2.25
	18	55	2.17	
Interval of 2 D.	20	50	1.97	2
	22	45	1.79	

In the old system the lenses are ground with a radius of curvature in Paris inches. The focal length is almost exactly the same in English inches as the radius of curvature is in French inches. The English inch = 25.4 mm.; the French inch = 27.07 mm.; 25.4 × 1.06 = 26.92.

In column three of the table the focus is given in English inches, as it is customary to compare the French lenses with the diopters by their focal length in English inches. A lens of 1 diopter has a focal length of 39.37 English inches. There is no lens in the old system which corresponds to it exactly. The nearest equivalent would be a lens of 40 inches.

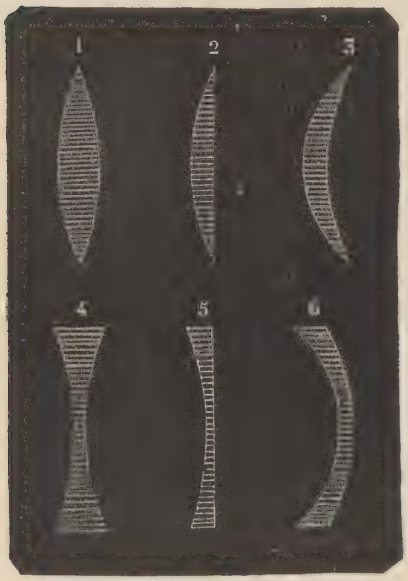


Fig. 16.—1. Biconvex lens. 2. Planoconvex lens. 3. Concavoconvex lens, convergent meniscus. 4. Biconcave lens. 5. Planoconcave lens. 6. Convexoconcave lens, divergent meniscus.

The lenses used for spectacles are spheric and cylindric.

Spheric Lenses.—A spheric lens is represented by a section of a sphere, or of two sections of a sphere placed together by their plane surfaces. Light passing through a spheric lens is refracted equally in all planes.

Cylindric Lenses.—A cylindric lens is a section of a cylinder parallel to its axis. Light passing through a cylindric lens is not refracted in a plane parallel to its axis, but in a plane perpendicular to the axis; rays are rendered convergent or divergent according as the cylinder is convex or concave (Figs. 17, 18).

Convex lenses are designated +; concave lenses, −.

Toric Lenses.—A solid developed by the revolution of a circle about any axis other than its diameter is known as a *torus*. A *toric lens* may be described as one which is cut from a toric surface by a plane parallel to its axis of development. The optical centering of such a lens requires that both its centers, the center of its circle and the center about which in its development the circle revolves, shall be on the axis of the system (W. S. Dennett). With the toric lens the angle of distinct view is increased, but a certain amount of astigmatism remains in the greater number of powers. To eliminate this astigmatism of oblique pencils of light M. von Rohr has developed a lens which it is claimed "reproduces any given definite point of an object as a distinct point in the image, that is, a lens which is corrected for astigmatism over the entire field of vision in all powers." To this lens the name *Punktal* has been given.

Combination of Lenses.—If two or more lenses are placed to-

gether, for example, + 2 diopters, + 3 diopters, and + 4 diopters, the combination forms a dioptric power equal to their sum—viz., 9 diopters;

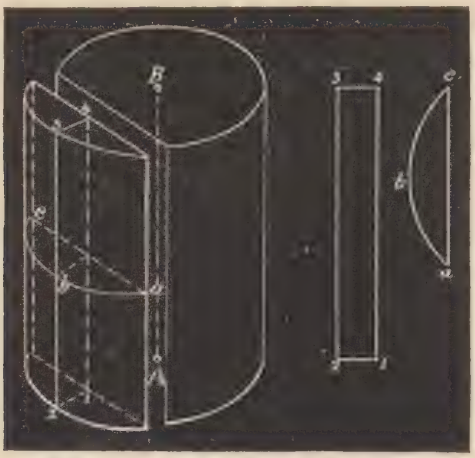


Fig. 17.—Convex cylindric lens, formed by a section of a cylinder parallel to its axis, which acts like a plane lens (*1*, *2*, *3*, *4*), in a direction parallel to the axis of the cylinder (*A*, *B*), and like a convex lens (*a*, *b*, *c*), in a direction perpendicular to the axis (Elschnig).

such a combination has, if composed of thin lenses, a focal distance of $\frac{100}{9}$ = 11 cm. If these lenses are placed at their focal distance from

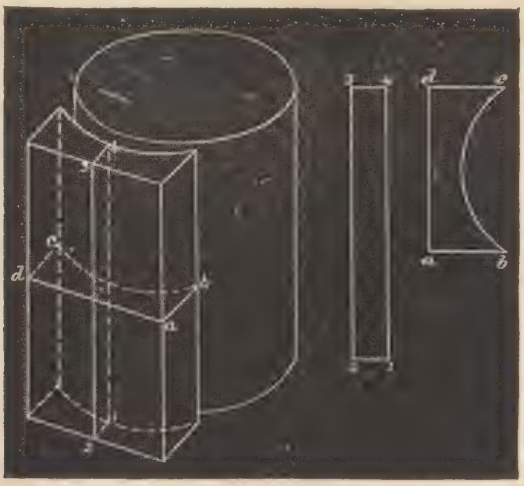


Fig. 18.—Concave cylindric lens, formed from a solid cylinder; in a plane parallel to the axis it acts like a plane lens (*1*, *2*, *3*, *4*), but in a plane perpendicular to the axis like a concave lens (*a*, *b*, *c*, *d*) (Elschnig).

an object, the rays coming from the object, after passing through the lenses, are parallel.

Two or more concave lenses placed together likewise produce a dioptric effect equal to their sum.

Combination of Convex and Concave Lenses.—If a concave and a convex lens of equal strength are placed together, they will neutralize each other so exactly that a distant object viewed through them will appear neither enlarged nor diminished, and there will be no prismatic deviation on gently shaking the lenses in a direction parallel to the surface.

Should they be unequal in strength, on shaking them an object (the edge of a wall or window frame is suitable) will be displaced toward the center of the lens if the concave is stronger, and away from the center if the convex is stronger. The value of the combination will be the difference between the strength of the two. For instance, a + 3 diopter and a − 2 diopter equal + 1 diopter; a + 2 diopter and a − 4 diopter = − 2 diopter.

A − 2-diopter lens gives to parallel rays a direction as if they came from a point 50 cm. away. Conversely, rays diverging from any near point may be represented by a concave lens, the principal focus of which equals that distance. Let rays, for example, diverge from a point 15 cm. away; they evidently are similar to parallel rays which have passed through a concave lens of 15 cm. local distance, $\frac{100}{15}$ = 6.66 diopters.

If it is desired to find the conjugate focal distance of any lens for rays which diverge from 15 cm., 6.66 should be subtracted from the dioptric power of the lens; the remainder gives a lens the focal distance of which is the conjugate desired. If it is desired to find the conjugate focal distance of a 12-diopter lens for rays which diverge from 15 cm., 6.66 should be subtracted from 12 = 5.33 diopters; 18.8 cm. is the conjugate focal distance.

Combination of Cylindric Lenses with Spheric Lenses.—A cylindric lens is curved only in the direction *perpendicular to its axis;* rays which enter the lens are refracted in this plane to the focus of the lens exactly as in the case of a spheric lens.

In the opposite direction, that is, *parallel to its axis*, the surface of a cylindric lens is flat; rays entering are not refracted in this plane, but pass through unchanged. The effect of a cylindric lens placed in front of the eye is to increase or diminish its refraction in the direction at right angles to its axis, but in the opposite direction the refractive power is unchanged (see Figs. 17, 18).

A convex 4-diopter cylindric lens, with its axis in a vertical direction (written + 4 D cyl., axis 90°), increases the refraction in the horizontal direction 4 diopters, but does not alter the refraction in the vertical direction. The horizontal plane is expressed by the term *horizontal meridian;* the vertical plane by the term *vertical meridian.*

A concave cylindric lens of 4 diopters, with its axis horizontal (written − 4 D cyl., axis 180°), diminishes the refraction of the vertical meridian 4 diopters, but does not affect the refraction of the horizontal meridian.

A convex lens of 3 diopters, combined with a convex cylindric lens of 2 diopters, with its axis vertical (written + 3 D ⌒ + 2 D cyl., axis 90°), adds to the horizontal meridian + 5 diopters, but to the vertical meridian only 3 diopters.

The combination of a convex spheric lens with a concave cylindric lens has the following effect: In the direction parallel to the axis of the cylinder the combination equals the full refraction of the spheric; in the direction at right angles to the axis of the cylinder the refraction is equal to the difference between the two lenses. If the convex spheric is stronger than the concave cylinder, the difference is still represented by a convex glass. For example, + 2 D sph., ⌒ — 1.50 D cyl., axis 180° = + 0.50 D sph., ⌒ + 1.50 D cyl., axis 90°, because + 2 D in the meridian of 180° is not diminished, but in the meridian of 90° it is reduced to + 0.50 D. Now, + 0.50 D sph. produces this amount of refraction at 90°, and supplies + 0.50 D of the requisite + 2 D at 180°, leaving + 1.50 D to be supplemented by a cylindric lens with its axis at 90°.

In place of writing + 2 D sph., ⌒ − 1.50 D cyl., axis 180°, a more simple expression would be + 0.50 D sph., ⌒ + 1.50 D cyl., axis 90°.

Where, however, the concave cylindric lens is stronger than the convex spheric, the difference is represented by a concave lens, thus + 3 D sph., ⌒ − 6.50 D cyl., axis 180°, signifies in the horizontal meridian convex 3 D, and in the vertical meridian concave 3.50 D. It is necessary to combine a convex with a concave lens in order to obtain this effect. The refractive power of this combination can be expressed in three different ways:

+ 3 D sph., ⌒ − 6.50 D cyl., axis 180°.
− 3.50 D sph., ⌒ + 6.50 D cyl., axis 90°.
+ 3 D cyl., axis 90°, ⌒ − 3.50 D cyl., axis 180°.

In the first combination + 3 D sph. gives the + 3 D necessary for the horizontal meridian, but increases the refraction of the vertical meridian 3 D instead of diminishing it; therefore the − 6.50 D cyl., axis 180°, expends 3 D of its refractive power in neutralizing the effect of the + 3 D sph., and with the remainder diminishes the refraction of the vertical meridian 3.50 D.

In the second combination, − 3.50 D sph., ⌒ + 6.50 D cyl., axis 90°, the concave spheric lens diminishes the refraction of the vertical meridian 3.50 D, but also diminishes the refraction of the horizontal meridian 3.50 D; as this already requires + 3 D, we must add + 3.50 D more to compensate for the concave spheric, making + 6.50 cyl., axis 90°.

In the third combination, + 3 D cyl., axis 90°, ⌒ − 3.50 D cyl., axis 180°, + 3 D cyl., axis 90° increases the refraction of the horizontal meridian without altering the refraction of the vertical meridian, and the − 3.50 D cyl., axis 180° diminishes the refraction of the vertical meridian without affecting the refraction of the horizontal.

With the combination of a convex spheric and cylindric lens, *e. g.*, + 3 D sph., ⌒ + 2 D cyl., axis 90°, a concave 0.50 D cylinder with its

axis at right angles to the axis of the convex cylinder, in this case at 180°, diminishes the refraction of the vertical meridian 0.50 D, the combination then equals + 2.50 D in the vertical meridian and + 5 D in the horizontal = + 2.50 D sph., ⌒ + 2.50 D cyl., axis 90°.

A convex cylinder + 0.50 D added to the same combination, with its axis at right angles to the axis of the first cylinder, that is, + 0.50 D cyl., axis 180° with + 3 D sph., ⌒ + 2 D cyl., axis 90°, increases the refraction in the vertical meridian + 0.50 D. The combination then equals + 3.50 D in the vertical meridian, + 5 D in the horizontal. This is obtained by + 3.50 D sph., ⌒ + 1.50 D cyl., axis 90°.

Visual Angle.—The apparent size of an object depends upon the size of the *visual angle.*

The visual angle is the angle formed by the lines drawn from the two extremities of an object to the nodal point of the eye. The *nodal point* of the eye is analogous to the optical center of a lens. It is situated 15 mm. in front of the retina and 7 mm. behind the cornea. Rays directed to this point pass through without deviation.

As the rays directed to the nodal point of the eye are not refracted, but continue the same course until they strike the retina, if lines are

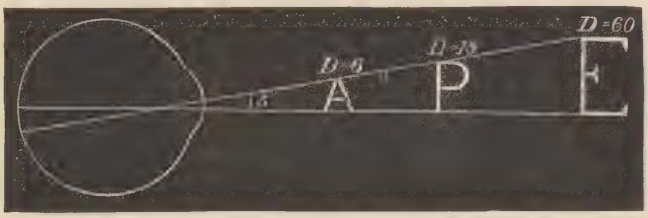


Fig. 19.—The visual angle.

drawn from the extremities of an object through the nodal point of the eye, and continued until they fall upon the retina, the size of the retinal image of the object is obtained.

The figure shows that the object, in order to subtend the same angle, must be larger the farther it is removed from the eye. The letter *A*, seen clearly at 6 meters, would have to be three times as large in order to be seen distinctly at 18 meters, and ten times as large in order to be seen clearly at 60 meters. The visual angle in the three cases remains the same.

Retinal Image in Emmetropia.—In the emmetropic eye the *nodal point* is situated 7 mm. behind the cornea and 15 mm. in front of the retina. The size of the retinal image is to the size of the object as the distance from the retina to the nodal point (15 mm.) is to the distance from the nodal point to the object. Therefore, if an object is situated at 1 meter distance (1000 mm.), its image will be $\frac{15}{1000}$ of the size of the object.

Retinal Image in Ametropia.—In the hyperopic eye, the axis of which is shorter than that of the emmetropic eye, the retina is situated

nearer the nodal point; the image is, therefore, smaller. In myopia the axis of the eye is longer; the retinal image is, therefore, larger.

Visual Acuteness; Limit of Perception.—An object 1 cm. in size, placed 1 meter distant from a normal emmetropic eye (that is, an eye without any error of refraction), is plainly visible. If this object is moved farther and farther away, it forms a progressively smaller visual angle, until a point is reached beyond which it cannot be perceived, owing to the diminutive size of the visual angle. The *limit of perception* has now been reached.

The angle which the object subtends at this distance from the eye represents the maximum *acuteness of vision*. An object twice the size would be seen distinctly at twice this distance. An object one-half the size could not be distinctly seen at more than half this distance. In general terms the size of the object denoting the acuteness of vision is always proportional to the distance.

Normal Acuteness of Vision.—Snellen determined the normal acuteness of vision to be the power of distinguishing letters subtending an angle of 5′. These letters are formed of strokes whose width is $\frac{1}{5}$ the size of each letter; consequently they are seen under an angle of only 1′. The openings in the letters and the spaces between contiguous strokes, as nearly as possible, are made to conform to the same angle.

Fig. 20.—Two of Snellen's test-types.

The relation of the size of the letter to the distance at which it should be discerned by a normal eye is expressed by twice the tangent of half the angle of 5′ = 0.001454. The size of a letter the perception of which constitutes normal vision at a given distance may be obtained by multiplying the distance by 0.001454. At the distance of 1 meter the size of this standard letter is 1.45 mm. (0.001454 × 1000 mm.). At a distance of 6 meters the size of the letter required is 8.7 mm. (1.454 × 6). The size of the retinal image of a standard letter of 6 meters = $\frac{15}{6000}$ of 8.7 = 0.02175 mm., and the strokes, or openings, being $\frac{1}{5}$ the size, have an image of 0.00435 mm. A large number of people, after correction of their ametropia, have a visual acuteness of 1.25 of normal, and, therefore, letters constructed on an angle of 4′ have been used for testing visual acuteness. The retinal images of the strokes of such letters are $\frac{4}{5}$ of 0.00435 = 0.00348 mm. The size of the cones of the macular region varies from 0.0033 to 0.0036 mm., showing a most interesting relation between the limit of perception and the anatomic structure of the retina.

ACCOMMODATION

Mechanism of Accommodation.—Inasmuch as the eye is inextensible, it cannot adapt itself for the perception of objects situated

at different distances by increasing the length of its axis, but only by increasing the refractive power of its lens. Rays diverging from near objects are thus brought to a focus at the same distance as the rays diverging from remote objects. This power the eye possesses of adapting its refraction for different distances is called *accommodation*, and the change required in its optical adjustment is effected by the ciliary muscle in the following manner: The ciliary muscle, which lies between the sclera and the ciliary processes, and which is attached posteriorly to the choroid tract by fibers known as the *tensor choroideæ*, contracts. This contraction draws forward the choroid and ciliary processes, to which is attached the suspensory ligament of the lens or zonula of Zinn. Hence the zonula is relaxed, and the tension which it has exerted on the lens capsule is removed. The crystalline lens, a soft and elastic body,

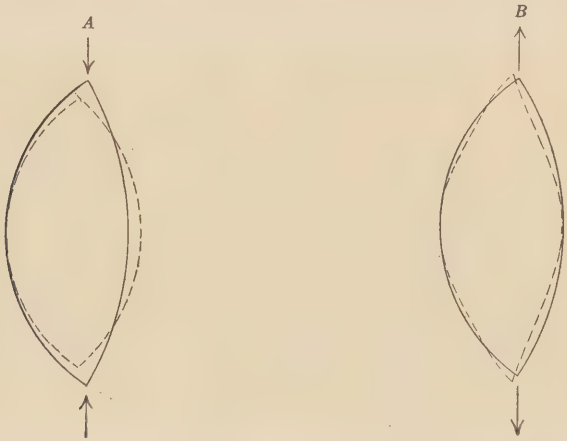


Fig. 21.—*A*, Accommodation according to Helmholtz. The dotted line represents the thicker form assumed by the lens when the traction of the zonula is diminished by the contraction of the ciliary muscle. *B*, Accommodation according to Tscherning. The unbroken lines show the lens at rest. The dotted lines show the change occurring during accommodation, supposed to be due to the traction of the zonula being increased by the contraction of the ciliary muscle (Cutler).

thus freed from compression, tends to assume a spheric shape, bulges forward, and becomes more convex. It has, in effect, added to its anterior surface another convex lens. As the ciliary muscle contracts more vigorously, this added convex lens becomes stronger. This is the Helmholtz theory, and attempts to disprove it have not been successful, as has been ably shown by C. Hess.

Tscherning holds a different view of the mechanism of accommodation, thus expressed by Colman Ward Cutler: Accommodation does not depend on a relaxation of the zonula of Zinn, but on its tension through the agency of the ciliary muscle, whereby the peripheral portion of the lens is flattened and the curve of the anterior surface, from an approximately spheric, approaches a hyperboloid form.

Karl Grossmann's careful investigation of a case of congenital aniridia indicates that the following changes occur during accommoda-

tion: The diameter of the lens equator becomes smaller; the antero-posterior diameter of the lens increases; the anterior pole of the lens moves forward, and its posterior pole backward; both the anterior and posterior surfaces of the lens form a lenticonus; the lens *in toto* moves upward and inward.

Exercise of Accommodation.—If an emmetropic individual wishes to see an object situated, for example, 25 cm. distant, he must exercise his power of accommodation to such a degree that in effect he adds to his crystalline lens another lens of 4 diopters—*i. e.*, one having a focal length of 25 cm. Rays diverging from 25 cm. are thus given a parallel direction and are brought to a focus on the retina by the original refractive power of the eye.

The degree of accommodation varies according to the distance of the object; it is not possible for an eye to be adapted for two different distances at one time. By means of the accommodation the eye is

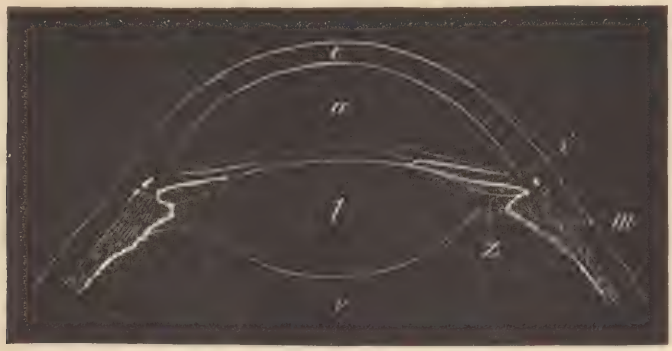


Fig. 22.—Increased convexity of the lens during accommodation. The solid white outline of the lens, *l*, shows its form when relaxed. The dotted line shows the increased curvature of the anterior surface during accommodation, and its advancement forward into the anterior chamber, *a*. *Z* is the suspensory ligament; *m*, the ciliary muscle; and *i*, the iris (Landolt).

adjusted for all distances between its farthest and nearest point of distinct vision.

The **far point** of an eye, *punctum remotum*, is the point from which come rays having the least divergence, or toward which go rays having the greatest convergence that allows their focusing on the retina. From this point rays are focused on the retina with the ciliary muscle entirely relaxed, the refraction of the eye being at its minimum, *R*. This point, or its distance from the eye, is designated *r*.

The **near point** of an eye, *punctum proximum*, or *p*, is the point from which come the most divergent rays that can be focused on the retina. These are focused with the ciliary muscle contracted to its fullest extent, and the eye in its condition of the maximum refraction, expressed by *P*. The space lying between *R* and *P* is called the *region of accommodation.*

The **range of accommodation,** likewise denominated the *power*

or *amplitude of accommodation*, is the difference between the refractive power of the eye accommodated for its far point and accommodated for its near point. This is expressed by A. $A = P - R$.

As the refractive power is the inverse of the focal distance, the refractive power of the eye, when accommodated for its far point r, is $R = \frac{1}{r}$. If we express the value of r in meters, we shall then have the refractive power of the eye expressed in diopters, a diopter being a lens of 1 meter focus. If $r = 1$ meter, $R = \frac{1}{1} = 1$ diopter $= 1$ D. If r is infinitely distant, $R \frac{1}{\infty} = 0$.

In the same manner $\frac{1}{p} = P$, the refractive power of the eye when accommodated for its nearest point. If we obtain the value of p in centimeters and wish to know how many diopters it equals, we must divide 100 by the number of centimeters equal to p. Let $P = 10$ cm., then $P = \frac{100}{10} = 10$ D. If p is expressed in fractions of a meter, we obtain the same result: by dividing 1 by the value of p, in meters, 10 cm. $= \frac{1}{10}$ of a meter. $P = \frac{1}{\frac{1}{10}} = 10$ D, or, in decimals, $1 \div 0.1$ meter $=$ 10 D—that is, in order to focus rays from 10 cm., we require 10 times as much accommodation as is necessary to focus rays from 1 meter, and since an eye adapted to a distance of 1 meter exerts 1 diopter of accommodation at a distance of $\frac{1}{10}$ meter, or 10 cm., it must exert 10 diopters of accommodation.

To find the range of accommodation we must first determine the far point. This is accomplished by means of test-letters held in front of the patient. If the patient has maximum acuteness of distant vision, r is infinite [when $R = \frac{1}{\infty} = 0$] or negative. If vision is less than normal at 6 meters, but is normal at 1.5 meters, $r = 1.5$ meters; R then $= \frac{1}{1.5} = 0.66$ D. If distant vision becomes or remains distinct when a convex glass of 2 D is placed before the eye, then $R = -2$ D; that is, the far point of such an eye is negative, a point behind the retina toward which rays converge. This condition is further discussed on page 123.

The *near point* is usually found by gradually approaching a card containing fine print until the nearest point from the eye at which it still remains distinct is reached. The distance of this point from the anterior surface of the cornea is measured. For this purpose large print may be reduced by photolithographing, so as to subtend the standard angle of 5′ at a distance of 25 cm. or less, and is usually arranged on suitably shaped cards. According to Duane, the best test object for practical purposes is a simple engraved line 0.2 mm. thick and 3 mm. long, which, when brought within the near point, blurs slightly and then doubles. In making his estimates he prefers to reckon from the

anterior focus of the eye—*i. e.*, from a point 13 mm. in front of the cornea.

The *formula for obtaining the range of accommodation* is $A = P - R$. If p is at 20 cm., $P = \frac{100}{20} = 5$ D, and r is at infinity, $R = 0$, then $A = P = 5$ D. This is the case in emmetropia.

If p is at 10 cm., $P = \frac{100}{10} = 10$ D, and r is at 25 cm., $R = \frac{100}{25} = 4$ D, then $A = 10$ D $- 4$ D $= 6$ D. This is the case in myopia of 4 D. P is greater than A.

If p is at 50 cm., $P = \frac{100}{50} = 2$ D, and r is negative, -25 cm. $R = \frac{100}{-25} = -4$ D. $A = 2 - (-4) = 2 + 4 = 6$ D. This is the range of accommodation in a hyperope of 4 D, and equals the sum of P and R.[1]

The near point is closer to the eye in young life, while the lens is soft; as age advances the lens becomes harder and the near point gradually recedes until, at about the age of seventy, the near point has reached infinity, and p and r then coincide, and there is no range of accommodation.

The *range of accommodation* is nearly constant for the same age, so that if p is nearer than it should be, myopia may be suspected, or if it is farther away than the average, hyperopia (Fig. 23). For this purpose the table given below is often used, which records the average of P in diopters and p in centimeters for the different ages. (Compare with Fig. 23.)

TABLE OF THE RANGE OF ACCOMMODATION.

10 years	14 diopters	p = 7	cm.
15 "	12 "	" = 8.3	"
20 "	10 "	" = 10	"
25 "	8.5 "	" = 12	"
30 "	7 "	" = 14	"
35 "	5.5 "	" = 18	"
40 "	4.5 "	" = 22	"
45 "	3.5 "	" = 28	"
50 "	2.5 "	" = 40	"
55 "	1.75 "	" = 55	"
60 "	1 "	" = 100	"
65 "	0.75 "	" = 133	"
70 "	0.25 "	" = 400	"
75 "	0 "	" = ∞	"

Duane and J. B. Thomas, in an attempt to determine the normal range of accommodation, have reached conclusions somewhat at variance with those of Donders, which are usually recorded. The accommodation in childhood and youth they found to be not so high as he states. The accommodation does not decrease year after year by any steady sweep, inasmuch as it may remain unchanged for years at some periods of life. After fifty-one the accommodation remains

[1] p refers to the distance of the near point in centimeters. P refers to the refractive power of the eye in accommodation for p. r refers to the distance of the far point. R refers to the refraction of the eye when accommodated for r.

nearly constant, diminishing only 0.50 D in ten years. Duane's researches are calculated to fix not only the mean range of accommodation, but also the upper and lower limits at each age.

Abnormal Accommodation.—Failure of accommodation due to age is termed *presbyopia* (see page 148). The chief anomalies of accommodation, adopting and quoting Duane's classification, are

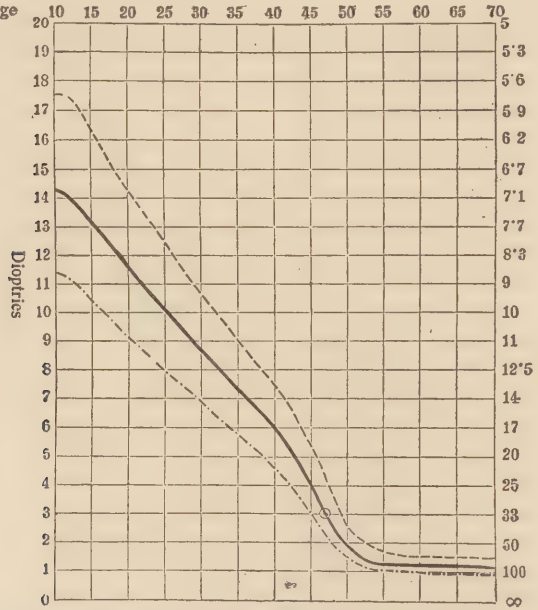


Fig. 23.—Range of accommodation at different ages. The numbers on the left hand give the strength of the lens, which placed before the emmetropic eye at a distance of 13 mm. from the apex of the cornea (*i. e.*, placed at the anterior focus of the eye), can replace the accommodation of the eye at the given age, and hence is equivalent to the accommodation, so far as regards the increase of refractivity in the eye which the latter produces. These numbers, therefore, give the range of accommodation of the eye. The right-hand numbers give the focal distances of these lenses (in centimeters). The distance of the near point from the apex of the cornea is found by adding to the focal strength of the lens its distance from the cornea (*i. e.*, 13 mm.). Obviously, no single observer can follow the progress of the range of accommodation from youth to age in one and the same individual. The progress of accommodation can, therefore, be found only by determining it in a large number of persons with normal eyes at different ages, and taking the mean of the observations. This is shown by the continuous line, which indicates the mean position of the near point (without reckoning in the 13 mm.) of an emmetropic eye at different ages. The upper and lower dotted lines give the least and the greatest distance of the near point that has been found in the individual cases. They accordingly show the limits within which the position of the near point can still be regarded as normal. The ring denotes the limit of presbyopia as conventionally set. (After Donders, amended and described by Duane.)

insufficiency of accommodation (see also p. 122), in which the accommodation is constantly below the lower limit; *ill-sustained accommodation*, in which accommodation is normal in amount, but soon gives out; *inertia of accommodation*, in which difficulty in changing from one accommodative state to the other is experienced; *inequality of accommodation* (see also page 150), in which the accommodation in the two eyes is not

the same; and *excessive accommodation*, in which the accommodation is persistently above the normal limit.

Insufficiency of accommodation, according to Duane, usually occurs, and it is not infrequent between the ages of eighteen and forty-five. Theoretically, it is due to undue rigidity of the lens or weakness of the ciliary muscle, and may be associated with an undue effort of convergence, that is, convergence excess (see page 555), but also and more frequently with convergence insufficiency (see page 555). The main symptom is *asthenopia*, and prominent causes are various toxemias due to tuberculosis, intestinal disorders, nasopharyngeal, tonsillar, and dental disease, hypopituitarism, neurasthenia, overwork, eye strain, and vascular hypertension. In the author's experience early arteriosclerosis is a common cause of this condition. Evidently the remedies are removal of the cause, suitable correcting glasses, with an addition for reading to correct the unnatural presbyopia, and, in the author's experience, the instillation of a weak eserin or pilocarpin solution, which, however, Duane has not found to be of much avail. Unequal accommodation, when non-pathologic in origin, may give rise to much discomfort, especially when the presbyopic age is approached or reached, and must be recognized in the correcting glasses (see also page 150).

Angle Gamma: Angle Alpha.—The eye, in looking at any object, is directed forward in such a manner that the image is formed on the *macula lutea*. The eye is now said to "fix" the object. A line drawn from the object thus fixed to the macula lutea is called the *visual line*, or *visual axis*.

The point about which the eye revolves, in order to be brought into this position, is called the *center of rotation*, and has its position 14 mm. back of the cornea. The line which connects the object with the center of rotation is designated the *line of fixation*.

The *optic axis* is an imaginary line passing through the center of the cornea and lens and the point of rotation to the posterior pole of the eye—*i. e.*, a point usually between the macula and optic papilla.

If the macula lutea coincided with the posterior extremity of the optic axis, the visual line, line of fixation, and optic axis would also coincide. Generally, this coincidence does not exist. In emmetropia and hyperopia the optic axis passes to the inner side of the macula lutea, and the visual line and line of fixation then form angles with the optic axis. In Fig. 24 A–A' is the optic axis passing through the center of the cornea, C, the nodal points of the eye, K'–K'', and the center of rotation, M. O–F is the visual line connecting the object, O, with the *fovea*, F. O–M is the line of fixation, drawn from O to the center of rotation, M. The eye, in order to fix O, has its optic axis, A–A', deviated outward. The angle formed by the line of fixation, O–M, with the optic axis A–A', is called the *angle gamma*, γ, or the angle formed by the visual line with the optic axis may be considered as the angle gamma.

The significance of this angle is that a person, while really fixing an object, seems to have a divergence of the visual lines—divergent squint. In estimating the degree of a divergent strabismus it is neces-

sary to consider the value of this angle. The amount of the angle gamma is usually 5°, but it may reach as much as 10°. When the anterior extremity of the visual line passes to the inner side of the optic axis, the angle gamma is positive, or +; this is the usual condition in emmetropia and hyperopia. The convergence of the visual line exceeds the convergence of the optic axis by the amount of this angle.

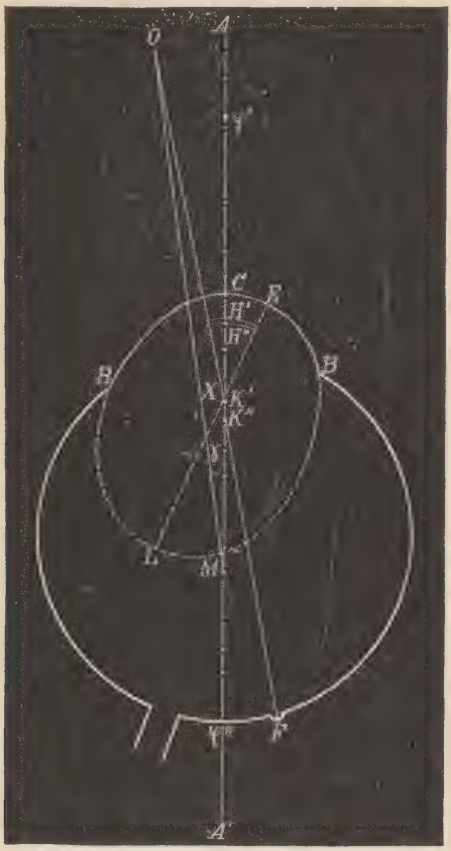


Fig. 24.—Angle alpha and angle gamma: *A–A'*, Optic axis; *O–F*, visual line; *O–M*, line of fixation; *E–L*, major axis of corneal ellipse. The line of fixation does not correspond with the optic axis, but forms the angle *O–M–A*, angle gamma nearly equal to the angle *O–X–A*, formed by the visual line with the optic axis. *O–X–A* may be considered as the angle gamma. The visual line does not pass through the summit of the corneal curve, *E*, but forms with the axis of the cornea, *E–L*, the angle *O–X–E*, the angle alpha (Landolt).

When the visual line coincides with the optic axis, there is no angle gamma. The visual line in high myopia sometimes passes to the outer side of the optic axis. The eyeball must then be deviated inward in order to fix on the object. This produces the effect of a convergent squint. It must be distinguished from squint; and if convergent strabismus also exists, the value of this angle must be deducted from

the apparent squint. In this latter form of the angle gamma, where the anterior extremity of the visual line passes to the outside of the optic axis, the angle is negative, or −. The convergence of the visual line is less than the convergence of the optic axis by the amount of this angle.

The amount of this angle may be measured by placing the patient before the perimeter as if his field were to be taken (see page 547). The eye is fixed on the central point, and a lighted candle is moved along the arc in a horizontal direction until its reflection is obtained from the portion of the cornea corresponding to the center of the pupil. The position of the candle may now be read from the arc in degrees, and represents the size of the angle gamma.

The apex of the cornea does not generally coincide with the center of the cornea, but is displaced laterally. The major axis of the corneal ellipse, represented in the figure by *E–L*, therefore forms an angle with the visual line. The *angle alpha* is the angle formed by the visual line with the major axis of the corneal ellipse. It is *positive* when the major axis of the cornea passes to the outer side of the visual line; if the corneal axis passes to the inner side of the visual line, the angle alpha is *negative*. In the figure the angle *O–X–A* is the *angle gamma;* the angle *O–X–E* is the *angle alpha.*

From what has been said it will be seen that the visual line is a secondary axis to the optical system of the eye. The oblique position of the refracting surfaces to the visual line may be the cause of an increased refraction in the horizontal meridian constituting astigmatism.

CONVERGENCE

In the visual act of one eye the sensation conveyed to the brain is projected outward over the same course by which it arrived—that is, the object is referred to a position in the field of vision which it actually occupies. If the projection outward of the images of the two eyes is such that they overlie each other, the person will have single vision; if, however, they are projected in different positions, double vision is the result.

The images are projected in different positions when they are not formed on *identical points* of the two retinas. The *fovea centralis* being the most sensitive portion of the retina, the eye is naturally so directed toward an object that the image is formed upon it. The eye is then said *to fix* the object. The foveæ of the two eyes are identical points, and images formed on them are projected outward so as to overlie or fuse into each other; points at a corresponding distance to the right of each fovea, or to the left, or upward or downward, are also identical, and images formed on them produce but a single impression. Objects in the field of vision to the right of the point of fixation form a retinal image to the left of the fovea. Objects to the left of the point of fixation form an image to the right of the fovea (see Figs. 229, 230). All images formed on the retina to the right of the fovea are projected outward to the left. Those formed on the left of the fovea are pro-

jected to the right; in the same way those formed on the upper part of the retina are projected downward, and those formed on the lower part of the retina are projected upward.

Fig. 25.—Meter angles of convergence (Landolt).

The eyeballs are separated laterally, on the average, 64 mm. in adult eyes. In looking at a distant object, if the axes of the eye are parallel, the images are formed on corresponding points of the retinas, but when the object is at some nearer point, the eyes must be turned inward in fixing the object, to compensate for their lateral separation. This function of the eyes is termed *convergence.*

The eyeball is rotated inward by the internal rectus muscle, so that its visual line is directed toward the object. This function is very closely associated with that of *accommodation;* one cannot act in any very great degree without the other also coming into play. The movement inward of the eye is measured by the angular deviation of the visual line, termed the *angle of convergence.*

The unit of convergence is the angle through which the visual axis moves to fix on a point 1 meter distant. This is termed *1-meter angle of convergence* (Nagel; Fig. 25). If the object fixed is only $\frac{1}{2}$ meter distant, the movement will be twice as great; it is then 2-meter angles. A point at $\frac{1}{3}$ meter would require 3-meter angles, and so on; 10-meter angles of convergence mean that the eye is directed to a point only $\frac{1}{10}$ meter distant.

Meter Angle.—In the figure, *O* and *O′* represent the centers of rotation of the two eyes; *O–O′* is the distance between these points, termed the interocular distance. It is measured by the distance between the pupils during fixation for remote objects. *O–M* is one-half this distance.

The line *C–M* is perpendicular to *O–O′*. When the object is situated on the line *C–M*, the convergence of each eye is equal. When the visual lines *J–O* and *J′–O′* are parallel, the angle of convergence is *nil;* when, however, the visual lines are directed to *C′*, 1 meter distant, *O–J* has deviated to *O–C′*. *J–O–C′* is the angle through which the visual line has moved to fix on *C′*. This is 1-meter angle of convergence.

C–M being parallel to *J–O*, *O–C′–M* is equal to *J–O–C′*.

In the right-angled triangle *O–C′–M*, *O–M* equals one-half the interocular distance.

O–C′ = the distance of the point of fixation.

$\frac{O\text{–}M}{O\text{–}C'}$ = the sine of the angle *O–C′–M*.

The average interocular distance is 64 mm. O–M = ½ of 64, or 32 mm. O–C' is 1 meter distant.

$\frac{O\text{–}M}{O\text{–}C'} = \frac{32}{1000} = 0.32$ = the sine of 1-meter angle. This corresponds to 1° 50′.

If the eye is directed to a point ½ meter distant, C'', the visual line will deviate twice as much—that is, it deviates 32 mm. at ½ meter distance. If the point of fixation is only $\frac{1}{10}$ meter distant, the amount of convergence will equal 10-meter angles.

To find the value of this in degrees we employ the same formula as above:

$\frac{O\text{–}M}{O\text{–}C^{10}}$ = sine of angle O–C^{10}–M. O–M = 32. O–C^{10} = $\frac{1}{10}$ meter = 100 mm.

$\frac{32}{100} = 0.32$, the sine of angle of convergence = 18° 40′.

The value of the meter angles in degrees is obtained very nearly by multiplying 1° 50′ by the number of meter angles. The value of the meter angle varies with the interocular distance, and as there is considerable difference in this distance, a separate calculation is necessary for each individual.

A more simple method of determining the value of the meter angle is to find its relation to the centrad. The centrad is a prism which deviates a ray the $\frac{1}{100}$ part of the radius, measured on the arc (see page 19). The deviation of the meter angle is measured on the sine. For the angles obtained, the sine and arc are almost equal.

One-meter angle equals a deviation of 32 mm. (the average distance between the centers of rotation of the eyes being 64 mm.) at 1 meter distance = 32 in 1000 mm., or 3.2 in 100 = 3.2 centrads. One centrad = 0.57295°; 3.2 centrads = 1° 50′. Ten-meter angles equal a deviation of 32 mm. in $\frac{1}{10}$ meter, 100 mm., 32 in 100, or 32 centrads = 18° 20′. A 32-centrad prism not only gives us the value of 10-meter angles of convergence, but, placed before the eye with the base inward, it takes the place of 10-meter angles of convergence, so that the eye, without any convergence, would see an object on the line C'–M, 10 cm. distant, as if it were situated at a remote distance.

The convergence becomes *greater* as the point of fixation approaches *nearer*. The number of meter angles is, therefore, inversely proportional to the distance expressed in meters. We thus designate the convergence in terms which indicate the same number of units of convergence as the diopters of accommodation necessary for the same distance. An emmetrope, in looking at an object 0.25 meter distant, would employ 4-meter angles of convergence and 4 diopters of accommodation.

The **amplitude of convergence** is the number of meter angles of convergence which the eyes can call into action. It is measured from the *far point* of *convergence* to the *near point* of *convergence*.

The far point of convergence is the point to which the visual lines are directed when the convergence is relaxed to its utmost; the near point of convergence is the point to which the visual lines are directed when the convergence is at its maximum. If in the minimum degree of convergence the visual lines are parallel, the far point of convergence will be at an infinite distance. Usually the visual lines actually diverge forward at the minimum of convergence, constituting an outward squint,

and converge by their posterior extremities toward a point behind the eyes. When this is the case, the far point and a portion of the amplitude of convergence are negative. In some cases, with the convergence relaxed to its fullest extent, the visual lines still deviate inward, constituting an internal squint. The convergence will in such a case always be entirely positive.

Relation between Accommodation and Convergence: Relative Accommodation.—While the two functions of convergence and accommodation, as has already been stated, are closely associated, there is still some independence of action. In other words, it is possible to accommodate several diopters without any convergence and to converge several meter-angles without accommodation. If the visual axes converge to a given point, the accommodation may be increased to a certain limit. The increased amount of accommodation exercised in these circumstances is measured by the ability to overcome concave glasses while the object still remains distinctly in view, and is denominated *the positive part of the relative accommodation.* It is also possible, while the visual lines converge for a given near point, to relax the accommodation from its association with that degree of convergence by placing convex glasses before the eyes, the object still remaining distinctly in view. This relatively diminished amount of accommodation is called *the negative part of the relative accommodation.* That convergence may be altered while the same effort of accommodation is maintained is demonstrable by placing a prism with its base inward before one eye, which then rotates outward, in order that the object may be seen singly, this object at the same time being perfectly distinct. Evidently the same effort of accommodation has been maintained, although the convergence of the visual axes is altered. At the far point of accommodation and convergence the accommodation has somewhat more play; at the near point, however, convergence has much the larger movement. The amplitude of convergence does not always diminish with age, as does the accommodation. Some persons, however, have a diminished convergence power or endurance, owing to changes in the ocular muscles similar in kind, though less in degree, to the senile changes which usually occur in other parts of the muscular system. Lucien Howe has designed an apparatus for the clinical measurement of the relative accommodation at the near point.

CHAPTER II

EXAMINATION OF THE PATIENT AND EXTERNAL EXAMINATION OF THE EYE; FUNCTIONAL TESTING

A SYSTEMATIC examination of each patient should be made in order to secure the preservation of careful records. For this purpose the following order of examination may be used:

Name and residence.

Age, sex, race, married, single, or widowed.

Family history: hereditary tendencies; general and ocular health of parents, brothers, sisters, etc.

Personal history: children, their general and ocular health; miscarriages; menopause; former illnesses; syphilis; gonorrhea; injuries.

Occupation: relation of work to present indisposition.

Habits: brain use; tobacco; alcohol; tea and coffee; narcotics; sexual.

Date and mode of onset and supposed cause of present trouble; outline of its course.

Organs of digestion: teeth; mouth; tongue; tonsils; stomach; intestines.

Organs of respiration: nose; accessory sinuses; throat; lungs.

Organs of circulation: heart; pulse; blood.

Kidneys: examination of urine.

Abdominal organs: liver; spleen.

Organs of generation; menses; leukorrhea; uterine disease; urethral and seminal vesicle and prostatic disease.

Wassermann test and tests for tuberculosis: fixation-complement test for gonorrhea.

Metabolic analysis.

Nervous system; intelligence; evidences of hysteria; hallucinations; sleep; vertigo; gait; station; tendon- and muscle-jerks; paralysis; tremor; pain; subjective sensations; convulsions; headaches and their position.

Eyes: previous attacks of inflammation; injuries; infections; ocular palsy or squint; amblyopia; previous use of glasses; ability to use eyes.

Direct inspection and examination of eyes: inspection of the skull and orbits (symmetry or asymmetry); ciliary borders; puncta lacrimalia; upper and lower culdesacs; conjunctivæ; caruncles; corneæ (oblique illumination and loupe); irides (mobility and color); anterior chambers (depth and character of contents); vision; accommodation; balance external eye muscles; adduction, abduction, sursumduction; position of eyes; mobility of globe; tonometer; tension; light-sense; color-sense; fields of vision; field of fixation; ophthalmoscope; ophthalmometer; retinoscope; test-lenses.

This schedule of examination must be modified to suit individual cases, as the patient presents trivial local lesions directly discoverable by inspection, or forms of disease requiring detailed study for their proper interpretation.

Direct Inspection of the Eye.—After the preliminary examination which the patient demands, the surgeon proceeds to the direct inspection of the eye. The surface of the lids should be examined for swollen

superficial veins, a common index of inflammation of the globe; their edges for inflammation, parasites, and misplaced cilia; the puncta for permeability, pressure at the same time being made over the lacrimal sac in order to express from it through the puncta any contained fluid; the upper and the lower conjunctival culdesac for accumulated secretion, granulations, and foreign bodies; the palpebral conjunctiva for hardened secretion in glands; the caruncles for swelling, attached foreign bodies, and irritation by incurved cilia; and the conjunctiva for the information to be derived from its blood-vessels.

In order to evert the lid, observe the following rule: Require the patient to turn the eye strongly downward, seize gently the central eyelashes of the upper lid between the index-finger and thumb of the left hand, draw the lid downward and away from the ball, place the point of the thumb of the right hand above the tarsal cartilage of the lid

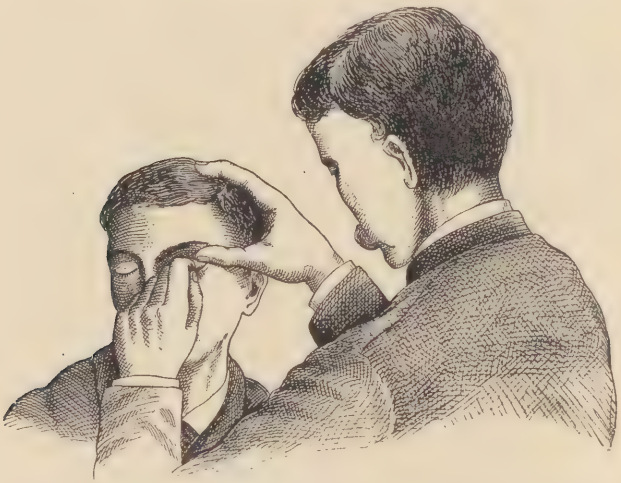


Fig. 26.—Position of hands in the act of everting the eyelid.

which is to be everted, the remaining fingers being steadied on the brow, and, by a quick movement, turn the edge of the lid over the point of the thumb, while this is simultaneously depressed. During the entire maneuver insist upon the downward direction of the patient's eyes; otherwise the lid cannot be turned without undue force and pain. When there are no lashes on the upper ciliary margin, the lower lid should be pushed beneath the edge of the upper in such a manner that it acts as a wedge on which the superior lid is then everted. In this manner the lid can be everted with the fingers of one hand. Care should be exercised to expose the tissues of the upper culdesac which lie beneath the folded lid.

The surgeon should inspect the skin of the face and forehead, examine the orbits by palpation, ascertain the action of the orbicularis by causing the patient to close his eyes as if in sleep, and study the length,

width, and symmetry of the palpebral fissures and the condition of the commissural angles.

Blood-vessels of the Conjunctiva.—In health only a few conspicuous blood-vessels are evident; in inflammation many more become visible. The arteries of the conjunctiva are derived from the palpebral and lacrimal branches of the ophthalmic; those of the episcleral tissue arise from the anterior ciliary branches of the ophthalmic, while the border of the cornea is surrounded by a plexus of capillary loops derived from the anterior ciliary vessels. This blood-supply may be conveniently divided, according to the late Mr. Nettleship, into three systems:

System I.—Posterior conjunctival vessels, whose congestion produces a bright red, velvety color, moving, on pressure of the eyelids, with the

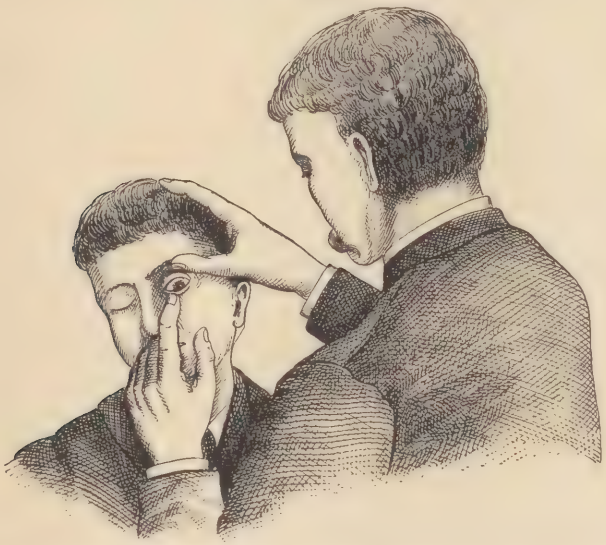


Fig. 27.—Eyelid everted for examination of its under surface and the upper part of globe.

shifting of the conjunctiva, usually associated with mucopurulent secretion, and indicating conjunctivitis. Conjunctival congestion is most intense at the fornix and in its neighborhood, and decreases as the corneal margin is approached.

System II.—Anterior ciliary vessels, composed of perforating and non-perforating arteries and veins. The perforating arteries, which supply the sclerotic, iris, and ciliary bodies, are the branches seen in health entering about 5 mm. from the corneal margin, their points of entrance, in dark-complexioned people, often being distinctly tinted.

The non-perforating (episcleral) branches, invisible in the normal eye, produce, when congested, a pink zone surrounding the cornea ("ciliary congestion," "circumcorneal zone"), not moving on pressure of the lids with the shifting of the conjunctiva, unassociated with

purulent discharge, and one indication of iritis. Ciliary congestion is most distinct around the corneal margin and lessens as the fornix is approached. As Haab remarks, the most congested circumcorneal zone is least involved in pure conjunctival congestion.

The perforating veins and their non-perforating (episcleral) twigs, when congested, create a zone of dusky hue, often a symptom of glaucoma, or appear in unequal, deep-seated patches of lilac or violaceous color, indicating cyclitis or scleritis.

System III.—Anterior conjunctival vessels and the plexus of capillaries surrounding the cornea, derived from anterior ciliary vessels through whose numerous small branches anastomosis between System I and II takes place. Their congestion produces a circle of bright-red injection, often partly on the cornea, a sign of inflammation of this membrane, and typified in the early vascular stages of interstitial keratitis (see page 267).

In addition to these three varieties of congestion, numerous departures are noticeable, making it impossible to separate the form and specify the individual system involved. In these types is found a definite local injection, as the leash of vessels passing to a corneal ulcer; or all the systems are commingled in a general inflammation. The blood-vessels of the conjunctiva can be well studied with a Zeiss corneal microscope.

Temperature of the Conjunctival Sac.—According to Silex, the temperature of the lower human conjunctival fold is 35.55° C. (95.99° F.)—*i. e.*, about 2° C. lower than that of the rectum. There is an average increase of 0.98° C. in inflamed eyes, the highest temperature being found in acute iritis. The temperature of the cornea is about 29° C.—84.2 F. (Leber).

Inspection of the cornea reveals inflammation, vascularization, ulceration, opacities, and foreign bodies. Slight irregularities are detected by placing the patient before a window, while the eyes are made to follow the uplifted finger held about 1 foot from the face, and moved in various directions. The image of the window-bars reflected from the cornea will be broken as it crosses the spot of inequality.

A more accurate method is to employ a *keratoscope* (Placido's disk). This instrument consists of a disk shaped like a target, upon which are drawn concentric black circles, a sight-hole being in the center. The patient is placed with his back to the window, while the surgeon holds the instrument in front of the eye, and, looking through the central aperture, observes the reflections of the circles from the cornea. If these are broken or distorted, the indications of irregularity in the surface are present.

Minute abrasions and ulcers may be found by dropping on the eye a concentrated alkaline solution of *fluorescein* (Gruebler's fluorescein, 2 per cent.; carbonate of soda, 3.5 per cent.), which colors green that portion of the cornea deprived of its epithelium, or in which the corneal epithelium is diseased, while the healthy epithelium remains unaffected. Epithelium in the immediate neighborhood of a

corneal ulcer, although apparently not involved in the process, will also take the stain, as pointed out by Benson. Cocain solution instilled prior to or after the application of fluorescein distinctly enhances its staining properties, and the epithelium of the cornea, which has been softened by repeated instillations of cocain, will take on the fluorescein stain. When the lesion is not very recent, or when it is covered with necrotic tissue, the coloration will be yellowish or yellowish green. This substance also reveals defects of the endothelium of the cornea, and E. von Hippel maintains that it produces a deep-seated coloration of the cornea only when the endothelium is absent or diseased (see page 334). C. A. Wood prefers a 2 per cent. solution of potassic fluorescide without the preliminary use of cocain. Toluidin-blue, as suggested by Veasey, and eosin may likewise be used as coloring agents.

The Width of the Cornea.—This may be measured approximately by holding before it a rule marked in millimeters, and noting the number of spaces its width occupies, or with Priestley Smith's *keratometer*, which consists of a scale situated between two planoconvex lenses. The average horizontal diameter of the normal cornea is 11.6 mm. (Priestley Smith).

The Sensibility of the Cornea.—This may be tested by gently touching the surface of this membrane with a wisp of cotton twisted to a fine point. If sensation is normal, the touch should be instantly followed by the reflex act of winking (*palpebral reflex*), although even when the cornea is insensitive closure of the lid may occur if the test-object comes into the field of the pupil. This is not due to contact, but represents the *retinal lid-closure reflex*. In organic anesthesia the *lacrimal* reflex is wanting, but is present in hysteric anesthesia. The opposite eye should always be tested as a control.

Oblique illumination is a method of examination by which the cornea, the anterior chamber, the iris, and, if the pupil is dilated, the lens and even the anterior layers of the vitreous may be studied. The surgeon places the patient 2 feet from the source of illumination, and focuses a beam of light with a 2- or 3-inch lens upon the cornea, at the same time observing the surface under examination through a lens of the same focal distance, held between the thumb and forefinger, the disengaged fingers being utilized to elevate the upper lid (Fig. 28).

The distance of the lens must be varied slightly, according as the cornea, iris, or crystalline lens is brought within its focus, the patient being required to look up, down, and to either side, while all the anterior surfaces and media of the eye are illuminated. In order to detect foreign bodies in the cornea, the light should be directed at an acute angle. If the posterior pole of the lens is to be examined, the light is thrown perpendicularly into the pupil, the surgeon placing his eye in the same direction without interfering with the light.

By this method minute abrasions, previously undetected foreign bodies, channels of old vessels, and other corneal changes may be examined. The character of the aqueous humor, the depth of the anterior chamber, the surface of the iris, the presence of synechiæ, small tumors,

atrophic fibers, and persisting pupillary membrane are evident, and, finally, opacities in the anterior capsule and axis of the lens are discoverable.

The Corneal Loupe.—This is a lens, properly mounted, by means of which the cornea is strongly magnified, and which should be employed with oblique illumination. A "corneal microscope" or a specially prepared lens of high power permits the study of minute changes in this membrane and in the iris, and is utilized for the examination of the traces of former vascularization, particularly after interstitial keratitis (see page 267), and by its help even the circulation of the blood in the vessels of a pannus may be studied. The Zeiss corneal microscope is particularly valuable in these respects.

Dr. Edward Jackson has designed a *binocular magnifying lens* which possesses material advantages. Berger has constructed on the same

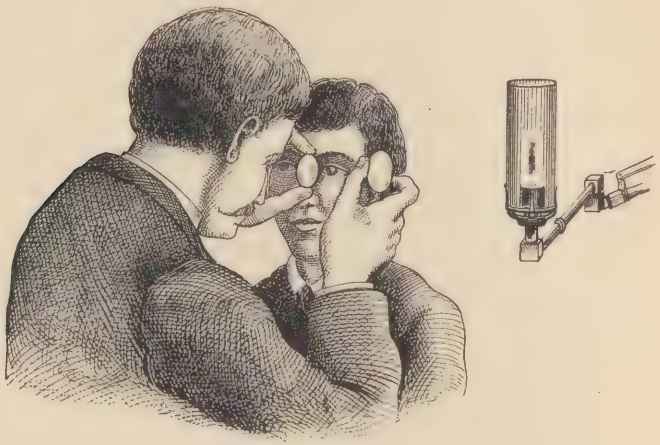


Fig. 28.—Method of oblique illumination.

principle a useful corneal loupe, the value of which is enhanced by the attachment of an electric lamp as designed by Shumway, and E. Treacher Collins has arranged a binocular magnifier mounted on a spectacle frame, similar to the Hess loupe, which also carries an electric lamp. The *Zeiss binocular magnifier* is a most useful instrument, its optical system being so constructed that plane surfaces appear flat to the eyes.

The Color of the Iris.—The color of the irides varies: blue and gray are the predominating hues in northern countries; brown occurs next in frequency; while the various admixtures produce yellow and green shades. Black irides are never seen; but dark irides, taking into account the whole population of the world, are of the most frequent occurrence. The color of the iris depends upon the amount and location of the pigment in it. Thus, if the coloring-matter does not exist in the stroma, but only in the posterior layer, the blue iris is evident, but if there is much pigment in the stroma, the brown or dark-brown

iris appears. The color of the iris of all newborn children is of a light grayish blue; the stromal pigment is developed subsequently.[1]

Slight differences in shade between the two irides are not uncommon; more rarely, even in health, the irides differ in color (chromatic asymmetry, *heterochromia iridis*), one being brown or greenish, the other blue or gray. Almost invariably, in cases of this sort, one iris corresponds in color with the irides of one parent, and the remaining iris with those of the other parent. Instead of uniform pigmentation, a single triangular patch or several irregular spots of dark color may appear upon one or both irides (piebald irides). This condition is sometimes temporary. Chromatic asymmetry, while perfectly compatible with health, has been observed in patients with neuropathic tendencies—chorea and epilepsy (Féré); the pupil of the blue eye may be smaller than that of the fellow eye; physiologic albuminuria may be present (T. Harrison Butler). In many instances there is liability to disease on the part of the lighter eye (cataract, cyclitis); indeed, the evidences of cyclitis, according to Fuchs, are nearly always present. For this complicated type T. Harrison Butler suggests the name *heterochromic cyclitis*. Examination reveals punctate keratitis, vitreous opacities, sometimes choroiditis and cataract. The opaque lens may be extracted and the results of operation are usually successful (Butler). This phenomenon may appear in several members of the same family. According to some authors the deficiency of pigment in heterochromia iridis is due to a lesion of the cervical sympathetic.

Discoloration from disease results in one iris being green, that of the yellow being blue, and indicates iritis or cyclitis; it is often an early symptom of inflammation of the iris, and should be looked for in every inflamed eye. When the dark segments seen in a piebald iris are small, they have been mistaken by incautious observers for foreign bodies.

The Pupil.—The size of the pupil in health varies with exposure to light and with accommodation and convergence. Changes in its width also depend upon the quantity of blood in the vessels of the iris, the elasticity of the iris-tissue, and certain mechanical conditions.

Under normal conditions the pupils—subject as they are to many influences—manifest certain fluctuations, amounting, according to Schwarz, to 0.3 mm., even when the chief factors are practically constant. The pupil is generally small in old age, in the newborn, and in eyes with hyperopic refraction; it is larger in youth, and in eyes with myopic refraction. Women are apt to have wider pupils than men. Exceptions to these statements are not infrequent, especially in so far as the relation of errors of refraction to pupil-width is concerned. Usually it is stated that the pupil is smaller in blue irides than in dark ones. Some recent investigations indicate that this is not the case. With the accommodation at rest, the diameter of the pupil varies in daylight from 2.44 to 5.82 mm., the average diameter being 4.14 mm. (Woinow). The position of the pupil is a little to the nasal side of the

[1] Ely records two dark irides in more than 1000 newborn children; one child was a negro.

center of the cornea, and, under similar illumination, the pupils should be round and of equal size (see also page 55). Slight inequality of the pupils is sometimes seen in healthy persons, and may be a congenital condition. Several instances of *cat-like pupils* have been observed in human eyes. In these circumstances the pupil in strong illumination takes on the form of a narrow elliptical slit (Greeff).

In addition to the factors already detailed which influence the size of the pupil, the *adaptation of the retina* to light must be taken into account, as Schirmer has shown. The pupil is exposed to clear daylight coming through a large window 1 meter distant, and the eye is permitted an adaptation of three minutes. Under such conditions a difference in width of 0.25 mm. has been determined. For the physiologic size of the pupil thus obtained Schwarz prefers the term *adapted width of the pupil.*

It is much to be regretted that the recorded variations in the diameter of the pupil are commonly imperfect, and that the loose statements, "pupils dilated," "pupils contracted," "pupils medium-sized," have crept into many reports.

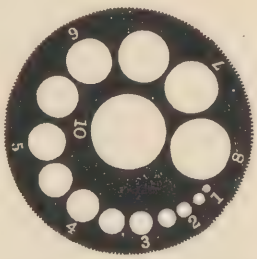


Fig. 29.—Simple pupillometer.

Measurement of the Pupil.—The pupil can be measured approximately by holding before it a rule, marked in millimeters, and noting the number of spaces its width occupies. The chief objection to this method is that the distance subtended on the rule is less than the diameter of the pupil, in proportion as the distance from the observer's eye is less to the rule than to the pupil (Jackson).

A great variety of instruments, known as *pupillometers*, have been devised for the accurate measurement of the width of the pupil. A simple and serviceable device is an instrument which consists of a scale of circles held close to the observed eye, the scale being rotated until that circle which matches the pupil in size is reached (Fig. 29). Priestley Smith's *keratometer* (see page 51) may be used for the same purpose. Haab's pupillometer, which consists of a number of black disks, varying from 1.5 to 8 mm. in diameter, arranged in a perpendicular row, with which the pupil is compared, is a useful instrument. Care must be taken that the hand using these instruments does not cast a shadow on the examined eye. Such examinations suffice for ordinary clinical work. For more exact determinations the photographic method of pupillometry is employed.

All examinations should be made under a uniformly strong light, and the character of light should be stated.

Mobility of the Iris.—Pupil-reflexes.—The mobility of the iris is tested to find the presence of attachments between the iris and the lens (synechiæ), or atrophy of the iris, or to ascertain the sensitiveness to light of the retina or visual center.

Variations in the size of the pupil depend upon variations in the contractility of the iris and upon alterations in the lumen of its blood-vessels. These pupillary movements[1] are controlled by the *pupil muscles* (see page 57) which are set in motion either by reflex stimuli, or by association with other voluntary or involuntary movements, that is, by *synkinesis*, to adopt Parsons' term. Such movements are often called *pupil-reactions* or *pupil-reflexes*. They are as follows:

1. The *direct light-reflex of the pupil*—that is, the contraction of the pupil obtained by illuminating the pupillary area. It may be tested as follows: The patient is placed before a window in diffuse daylight, and one eye is carefully excluded. He is directed to look into the distance with the exposed eye, which is then shaded, and, if it is normal, a considerable dilatation of the pupil will occur. On removing the covering hand or card, contraction to the same size as that which existed before the test was applied takes place. The test may also be conducted in the following manner: The patient is seated as before described, and *both* eyes, which gaze steadily in the direction of the light, are covered with the examiner's hand or a card, and after a few seconds the cover is removed from one eye and the initial width and the rapidity and completeness of the contraction of the exposed pupil is observed. The same procedure is repeated with the other eye. Again, inasmuch as a properly lighted window is not always available, the test should be made with artificial illumination. The patient is seated in a dark room in front of the source of illumination (Argand burner, Welsbach light, lamp, or electric light), and looks into distance. Convergence and accommodation are relaxed, and the diameter of the pupil is measured with a pupillometer. Next, light is reflected into the eye with the ophthalmoscope mirror and the pupil reaction noted. Finally, the patient is required to face the light and the pupil area is subjected to oblique illumination (see page 51) and the effects observed. This is the method recommended by Bach. Even more satisfactory results are obtained if the source of illumination is a narrow beam of electric light obtained, for example, from a Würdemann transilluminator (see page 363) upon which a cap containing a small condensing lens is fitted, as in the model designed by Veasey.

2. The *consensual light-reflex*, or *indirect reflex action of the pupil*—that is, the contraction of the pupil of one eye, which is evident when the pupillary area of the opposite eye is illuminated. The test is made as follows: One eye is completely excluded from the source of illumination, and the other shaded in such a manner that the pupil can be observed

[1] This term is so well established and so commonly employed by clinicians that, in spite of the objections to it which have been urged, it should be retained.

beneath the cover. The completely excluded eye is now uncovered and the light directed into its pupil, the reaction which occurs in the shaded pupil being at the same time observed. Although the pupil of one eye acts under normal conditions in unison with its fellow, the direct and indirect reactions are not equal in intensity. According to Bach, the direct reaction to light is greater than the consensual. The statement, often made, that in normal eyes the pupils should be equal, not only with both eyes open but with one eye shaded, is not strictly correct, and usually the difference in width may be demonstrated by allowing for some seconds the stronger illumination to fall on one pupil (Bach).

3. The *accommodation- and convergence-reaction*, called also the *associated action of the pupil*, or the *accommodation synkinesis*—that is, the contraction of the pupil which takes place when the visual axes converge upon a near point. Usually the test is made as follows: The patient is required to look into distance and then quickly to direct his eyes at a near object—for example, the point of a pencil held at a distance of about 10 cm. Under normal conditions a contraction of the pupils will occur—that is, the sphincter of the iris contracts in association with the ciliary muscle and the internal recti. Bach's procedure is the following: The patient, seated facing a wall between two windows, is required to observe for twenty seconds a small white button placed 50 cm. from his eyes. The button is then gradually approached. No change in the pupil is observed until the object reaches a distance of 40 cm. from the eye, as it is usually gradual at first. When a distance of 20–15 cm. is reached the contraction is stronger and may occur suddenly, associated with a strong convergence impulse. The amplitude of contraction, which is less marked than that which follows the action of light, varies between 0.25 and 0.75 mm.; exceptionally it is greater. Generally, it is less marked in old than in young persons. Refraction anomalies, according to Bach, produce no marked difference in the degree of contraction during convergence, except that in high myopia the reaction is sometimes delayed and less in amplitude. The associated movement of the pupil is much more closely connected with convergence than with accommodation; indeed, it is chiefly due to the impulse of convergence. If, experimentally, accommodation and convergence are dissociated, accommodation may take place without pupil contraction, but convergence cannot occur without contraction of the pupil (Swanzy and Werner).

4. The *sensory reflex of the pupil*, sometimes called the *skin-reflex*, or the *pain-reaction*—that is, a slight dilatation of the pupil which occurs on stimulating sensory nerves. It may be tested by pinching the skin of the neck, or, better, by applying to it a faradic brush.

5. The *cerebral cortex reflex of the pupil* is thus described by its discoverer, Dr. Haab: "If in a room illuminated only by a lamp or candle-flame, the light is placed so that it will shine laterally into a person's eyes while they look directly forward into the darkness, a marked contraction of both pupils takes place whenever the attention is directed toward the light, with no change in the position of the eyes. As long

as the attention is directed to the light and fixation of the eyes on the dark wall is maintained the pupils remain contracted, but as soon as the attention is transferred to the point of fixation they dilate, although the quantity of light entering the eye has remained constant and all movements of accommodation and convergence are excluded."

The clinical significance of this reflex has not been ascertained, although Haab believes it may have some important bearing on the theory of attention, and that it should be investigated in all patients submitted to neurologic examination.

The observation of Piltz that in some persons the pupils contract or dilate when they call up a vivid mental picture of a bright or dark object has given rise to the term *imagination reflex of the pupils.*

6. The *palpebral (lid-closure) reflex of the pupil,* also denominated the orbicularis pupillary reaction, the Gifford-Galassi reflex, and the Westphal-Piltz reaction, was discovered by Von Graefe. It consists of a contraction of the pupil which occurs when a forcible effort is made to close the lids. It has been explained by assuming that an associated stimulation of the sphincter nucleus takes place during closure of the lid, or that it is due to the mechanical effect produced by strong *contraction* of the orbicularis.

When a pupil has been contracted under the influence of light, convergence, or accommodation, and the stimulus is withdrawn, the pupil will return to the size it had before the stimulus was applied, if the conditions remain the same. This return or relaxation has been called by Walter Jessop *the dilatation- or relaxation-reflex of the pupil.*

Innervation of the Iris and Explanation of the Pupil-reflexes.—The muscular tissue of the iris is divided into the *sphincter pupillæ,* a well-marked circular band of involuntary muscle surrounding the pupillary margin of the iris, and certain radially placed fibers, much less clearly marked, situated near the posterior surface, called the *dilatator pupillæ.*[1] These two muscles are called the pupillary muscles, and each has a separate and independent motor-nerve supply which constitute the *miotic* and *mydriatic* nerves.

The third (oculomotor) nerve innervates the sphincter of the pupil, and contains the pupillo-constricting fibers which arise from its nucleus in the aqueduct of Sylvius. From this point the fibers proceed in the main trunk of the nerve to the orbit, and pass into the branch which supplies the inferior oblique, which they leave by way of the twig which constitutes the short root of the ciliary ganglion,[2] and finally arrive at the sphincter by the short ciliary nerves which penetrate the sclerotic around the optic nerve, and pass forward in the choroid and ciliary body to their destination in the iris. This line of communication between the nucleus of the third nerve and the sphincter of the pupil is

[1] The existence of a dilator muscle in the iris is denied by some authors, but the combined anatomic and physiologic evidence of its presence seems to be conclusive.

[2] As Langley and Anderson have shown, there is a cell-station in the ciliary ganglion. The root-fibers which belong to the oculomotor end in the ganglion, and with the cells of the ciliary ganglion a new neuron begins for the fibers which pass to the ciliary muscle and the sphincter of the pupil.

called the *miotic* tract or *efferent* path, and is also known as the *centrifugal* pathway of the pupil reflex. Stimulation of it produces contraction of the pupil; section of it, moderate dilatation.

The cervical sympathetic innervates the dilatator of the pupil. The dilatator tract proceeds from a center in the medulla (or from a

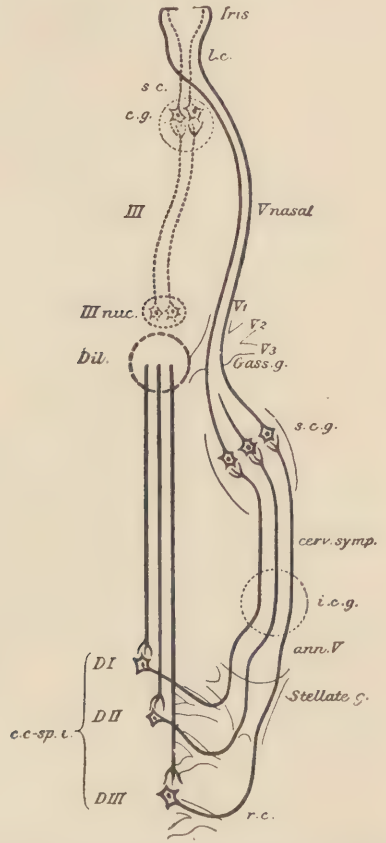


Fig. 30.—Diagram of the efferent pupillary paths. Dotted lines, pupilloconstrictor: *III nuc.*, nucleus of third nerve; *c. g.*, ciliary ganglion; *s. c.*, short ciliary nerves. Solid lines, pupillodilatator: *Dil.*, hypothetical dilatator center in the medulla; *c.c-sp. i.*, Budge's centrum ciliospinale inferius; *DI, DII, DIII*, first, second, and third dorsal nerves; *r. c.*, ramus communicans; *Stellate g.*, stellate ganglion; *ann. V*, annulus of Vieussens; *i. c. g.*, inferior cervical ganglion; *cerv. symp.*, cervical sympathetic; *s. c. g.*, superior cervical ganglion; *Gass. g.*, Gasserian ganglion; *V1, V2, V3*, first, second, and third divisions of the fifth nerve; *V nasal*, nasal branch of the ophthalmic (first) division of the fifth nerve; *l. c.*, long ciliary nerves. (Description and diagram from J. Herbert Parsons.)

point in the aqueduct) into the lateral columns of the spinal cord as far as the third dorsal nerve. The pupillodilating fibers leave the cord by the ventral roots of the first, second, and third dorsal nerves and follow their communicating branches to the superior cervical ganglion. They pass upward in the ascending or carotid branch of the first cervical ganglion and arrive at the plexus around the internal

carotid and the Gasserian ganglion. They reach the eyeball through the nasal branch of the ophthalmic nerve and its long ciliary nerves which perforate the sclerotic, and are distributed to the ciliary muscle and iris. The tract just described is called the *mydriatic* tract. Stimulation of it causes dilatation of the pupil; section of it, moderate contraction (Fig. 30).

Inasmuch as the iris is not under the control of the will, the contraction of the pupil which occurs when the eye is exposed to the source of light in the manner described is a reflex—that is, its motor nerves are excited to action indirectly by the reflex stimulus of light. This light reflex is under the control of the constrictor center, which the stimuli reach by passing along a tract which is known as the *afferent pathway*, the exact course of which is as yet uncertain, but it is probably somewhat as follows: The fibers of the pupil-reflex tract begin in the retina and arise from all parts of it and proceed in the optic nerves, and are in all probability to be histologically differentiated from those which are concerned with vision.[1] In the chiasm these pupillary fibers undergo partial decussation and enter the optic tracts, which they leave just in advance of the external (lateral) geniculate body, and reach the third nerve nucleus. From a special part of this nucleus, probably the small-celled median nuclei, the pupilloconstrictor fibers arise and reach the sphincter of the iris, constituting the efferent, miotic, or centrifugal pathway already described[2] (Fig. 31).

The direct light reflex of the pupil is the result of an active constrictor effect, the stimuli passing along the afferent pathway to the sphincter center in the third nucleus, and from there by the efferent pathway to the termination of the miotic fibers in the iris.

The consensual or indirect light reflex of the pupil occurs because the stimulus passes to the opposite eye, either by reason of the decussation of the fibers in the chiasm or because of its transference from one nucleus to the other.

The sensory reflex of the pupil is a dilator reflex called into existence by various sensory stimuli. According to Parsons, it is due in part to augmentation of the dilator tone through the sympathetic, and in part to inhibition of the constrictor tone.

The convergence and accommodation reaction of the pupil is not a reflex, but an associated movement, and has been ascribed to the effect of a stimulus which reaches the convergence center in the third

[1] According to some observers (Hess) the portion of the retina which receives the light rays, giving origin to the pupil-reflexes, is confined to a small central area with a radius of about 3 mm.

[2] The path by which the pupillary fibers leave the optic tract to reach the third nucleus, in the language of Parsons, is as yet conjectural. This author, referring to the pupillo-constrictor path, thinks it is probable that the fibers pass through the superior brachium of the quadrigeminal body to the superior colliculus, there making new connections with the cells which convey the impulses to the third nucleus of the same and also the opposite side. Von Hippel states that after the pupil fibers leave a tract in advance of the external geniculate body, they run up and in toward the median line, and as they enter the white substance of the corpora quadrigemina, they radiate, part of them going to the roof, and another part, under the aqueduct, toward the sphincter nucleus. It is probable that there is a connection between the two sphincter nuclei over the median line through the ganglion-cell processes.

nucleus, and is diffused to the cells which innervate the ciliary muscle. According to Schwarz, it is possible that a single cerebral impulse to accommodate both eyes to near vision stimulates simultaneously the nuclei which regulate convergence, accommodation, and pupil contraction.

The cerebral cortical reflex or, better, reaction of the pupil, is of complex nature and results from psychic stimuli. The explanation of the lid-closure reflex of the pupil has been given (see page 57).

Not only may constriction of the iris, and therefore contraction of the pupil, be due to contraction of the constrictor (sphincter) muscle, but it also may be caused by relaxation of the dilatator muscle and dila-

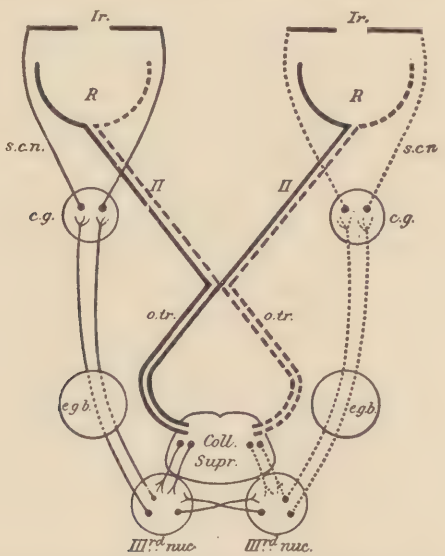


Fig. 31.—Diagram of the afferent and efferent pupillary paths for light stimuli. Afferent paths from left sides of retinæ, thick solid lines; afferent paths from right sides of retinæ, thick dotted lines; efferent paths of left eye, thin solid lines; efferent paths of right eye, thin dotted lines: *Ir.*, iris; *R*, retina; *II*, optic nerve; *o. tr.*, optic tract; *Coll. Supr.*, colliculus superior or anterior corpus quadrigeminum; *IIIrd. nuc.*, nucleus of third nerve; *e. g. b.*, external geniculate body; *c. g.*, ciliary ganglion; *s. c. n.*, short ciliary nerves. (Description and diagram from J. Herbert Parsons.)

tation of the blood-vessels of the iris. As before stated, the evidence strongly indicates that the light reflex is an active constrictor effect, although some writers maintain that it should be explained by an inhibitory dilatator influence. Instead of locating the center for the light reflex of the pupil in the small cells which occupy the median part of the third nucleus, Marina has placed it in the ciliary ganglion. Bach has described an inhibitory constrictor and an inhibitory dilator center in the spinal end of the floor of the fourth ventricle, and, according to him, irritation of these centers will lead either to dilatation or contraction of the pupil. The presence of these centers is denied by a number of observers.

Not only may dilatation of the pupil be due to contraction of the dilatator muscle (dilatator pupillæ), but may also be caused by relaxation of the constrictor (sphincter) muscle and to constriction of the blood-vessels in the iris. The dilatator pathway has been described (see page 58). Budge and Waller believed that the origin of the pupil-dilating fibers should be referred to the spinal cord in a region between the exits of the sixth cervical and fourth dorsal or thoracic nerve (probably opposite the seventh cervical and first thoracic), which is known as *Budge's ciliospinal center* (see Fig. 30). Although certain clinical and experimental evidence is in favor of this center, its existence has not been proved. Not only is the sympathetic pathway of the pupils concerned with maintaining a certain tone in the dilatator muscle of the iris, but it is also capable of being actively awakened by various sensory stimuli.

Dilatation of the pupil occurs in glaucoma, in optic-nerve atrophy, in orbital disease, and under the influence of mydriatics. It is further seen in fright, emotion, in deep inspiration or expiration, anemia, in depressed nervous tone, aortic insufficiency, cutaneous stimulation (*skin-reflex*), and irritation of the cervical sympathetic. If paralysis of accommodation is associated with dilatation of the pupil, objects may appear smaller than normal (*micropsia*).

In diseases of the nervous system, dilatation of the pupil, when of cerebral origin, indicates extensive lesion; when of spinal origin, irritation of the part (McEwen). Systematic writers have divided dilatation into *irritation mydriasis*, caused by irritation of the pupil-dilating center or fibers, and *paralytic mydriasis* (iridoplegia), caused by paralysis of the pupil-contracting center or fibers.

In *irritation* or *spastic mydriasis* the pupil may be moderately or widely dilated. It reacts somewhat to light, accommodation, and convergence if the dilatation is not extreme; but if it is, these reactions may be lacking. Cocain usually produces no further dilatation of such a pupil, nor is it readily contracted by pilocarpin, and sometimes not at all. It is seen in hyperemia and irritation of the cervical part of the spinal cord, in spinal meningitis, in tumor of the cord, sometimes in tumor of the cerebrum, in acute mania, and in early tabes dorsalis and paretic dementia. Spasmodic mydriasis has been observed in a healthy subject (Cramer).

In *paralytic mydriasis* (*sphincter paralysis*) the pupil is dilated, but not necessarily *ad maximum*. It does not react to light, accommodation, and convergence, and the condition is sometimes described as *pupillary rigidity*. If there is only paresis and not paralysis of the sphincter, a sluggish reaction to light, accommodation, and convergence may be obtained. Cocain still further dilates a pupil of this character, and it is contracted by the action of pilocarpin.

Paralytic mydriasis may be caused by a lesion in the sphincter, the sphincter nucleus, or the centrifugal tract. It is seen in disease of the base of the brain affecting the third nerve or its nucleus, in pressure on the cerebrum great in degree, in late stages of meningitis, in edema of

the cortex, in cerebral softening, in hemorrhage of the centrum ovale, and cerebral peduncles.

In *medicinal mydriasis—i. e.*, one caused by atropin or a similar drug or by certain toxins—there is paralysis of accommodation, and the pupil is unaffected by pilocarpin.

Contraction of the pupil (*miosis*) appears in congestions of the iris, in traumatisms of the iris (traumatic miosis), in certain fevers, in plethora, venous obstruction, mitral disease, pulmonary congestion, paralysis of the sympathetic, and under the influence of miotics. During sleep the pupils are contracted. If spasm of accommodation is associated with contraction of the pupil, objects may appear larger than normal (*macropsia*).

If the miosis is of cerebral origin, it indicates an early irritative stage of the affection (meningitis, etc.); if of spinal origin, a depression, paralysis, or even destruction of the part (McEwen).

Systematic writers have divided contraction of the pupil into *irritation miosis*, caused by irritation of the pupil-contracting center or fibers, and *paralytic miosis*, caused by paralysis of the pupil-dilating center or fiber. The same factors which cause miosis may cause mydriasis, the determining factor being the degree and the duration of the lesion.

In *irritation* or *spastic miosis* the pupil is contracted, in medium degree if one etiologic factor is active, *ad maximum* if both are concerned. Such a pupil dilates little or not at all in the dark, and usually is unaffected by the action of light. It is readily dilated by a mydriatic (atropin), and still further contracted by pilocarpin. The active lesion may reside in the iris, in the sphincter nucleus, or in the centrifugal pathway. According to some authors spastic miosis may be indirectly caused by failure of the inhibitory influences to act on the sphincter nucleus (Schwarz).

Irritation or spastic miosis may be caused by inflammatory affections of the base of the brain and the meninges in their early stages, by brain abscess, by beginning sinus disease, in the early period of cerebral neoplasms, in small hemorrhages in the cerebellum, at the onset of cerebral apoplexy, and in apoplexy of the pons. It is also seen in hysteria, at the beginning of epileptic attacks, in certain toxemias, in tobacco amblyopia, and under the influence of long-sustained efforts of accommodation.

In *paralytic miosis* (*dilatator paralysis*) the pupil is contracted, but its motility is preserved in that it reacts to light and the impulse of convergence. In the dark it dilates, but less perfectly than a normal pupil. Such a pupil is dilated by mydriatics, but only partially; it is contracted still further by miotics.

Paralytic miosis may be caused by lesions in the cord above the dorsal vertebra, and is especially noteworthy in tabes dorsalis (*spinal miosis*). It is also seen in paralysis of the insane, pseudodementia paralytica of syphilitic origin, in some forms of bulbar paralysis, and in some varieties of multiple neuritis (Mills). It is caused also by injury to the cervical sympathetic, or by pressure, for example, from enlarged cervical glands

or an aneurysm. If the cervical sympathetic is paralyzed, with the miosis there is enophthalmos and ptosis (*sympathetic ptosis*).

A pupil which does not react, either directly or indirectly (consensually), to the influence of light, but contracts promptly on convergence of the visual axes, exhibits the condition to which the term *reflex inactivity* or *immobility of the pupil* is applied, and which also is known as the *Argyll Robertson pupil*. Usually the condition is bilateral, but unilateral reflex inactivity or immobility is also seen, and even where the failure of light reaction is bilateral, one pupil may be smaller than the other, although both are miotic pupils. Sometimes the same reflex immobility is present when the pupils are dilated. Frequently the affected pupils are not round, but slightly oval or pointed. The seat of the lesion in these circumstances is not certainly known. It has been placed in the fibers which pass from the proximal end of the optic nerve to the oculomotor nuclei by some authors, and by others is considered to be nuclear. Bach believes that it may be located in the spinal end of the sinus rhomboidalis, and Marina and Lafon place the causal lesion in the ciliary ganglion.

Reflex immobile or inactive pupils are especially noteworthy in tabes dorsalis and paretic dementia, and, as is well known, may precede the general signs of these diseases by many years. They also occur in syphilis, but have also been observed in non-syphilitic affections and in alcoholic neuritis. If miosis is present it has been attributed to a sympathetic affection and to tonic contraction of the sphincter, but Bach thinks that it depends upon an irritation of the reflex inhibitory center which he believes he has discovered. According to the same author the Argyll Robertson pupil may remain unilateral for years, and exist as an isolated symptom.

The reverse of the Argyll Robertson symptom has been observed, that is to say, the pupil reacts to light, but fails to react to convergence, and has been ascribed to disease in a special part of the oculomotor nucleus.

Unilateral reflex iridoplegia, or a condition in which one pupil is unaffected by varying degrees of illumination of both eyes, but reacts to accommodation, while the pupil of the other eye responds to a separate light stimulus of either eye, and which is seen in tabes dorsalis and syphilitic cases, should be distinguished from *unilateral reflex blindness*, caused, for example, by interruption of the conducting power of one optic nerve. In unilateral reflex blindness illumination of the pupil area on that side fails to elicit either the direct or the indirect pupil-reflex.

Convergence Anomalies of the Pupils.—As already noted, in complete rigidity of the pupil, such as occurs with total paralysis of the sphincter, there is no convergence reaction, but it is conceivable, as Schwarz points out, that a common disturbance of convergence reaction and light reaction, due to interruption of the corresponding pathways leading to the sphincter nucleus, may occur without paralysis of the sphincter.

Occasionally a pupil which is inactive to light stimulus, but which contracts on convergence, will remain in this contracted condition for a considerable length of time before it slowly returns to its original size. This phenomenon has been called the *myotonic pupil movement* by Sänger, and *neurotonic convergence reaction* by Piltz. It has been noted in tabes dorsalis, in multiple sclerosis, migraine, and alcoholism. Failure of the convergence reaction of the pupil, unassociated with disturbances of the light reflex, although rare, may occur. It may be complete or incomplete, and the convergence rigidity may be associated with paralysis of accommodation, or this may be absent.

The *palpebral reflex of the pupil*, according to Bach, may be seen in normal eyes, and is perhaps accountable for some of the contradictory observations which have been made on the pupil reflexes. If there is sphincter paralysis it is abolished, but sometimes it appears, although light reaction and convergence reaction are absent, when it must be assumed that the sphincter itself is not paralyzed, or, at least, not completely disabled.

Unequal pupils (*anisocoria*) are rarely seen in health. If there is recent wide dilatation of one pupil and no disease of the eye, the instillation of a mydriatic may be suspected. Unequal pupils occur in eyes with widely dissimilar refraction, if one eye is blind, in aneurysm, pulmonary tuberculosis, dental disease, sinusitis, traumatism, and in diseases of the nervous system. If the disease is cerebral, the inequality denotes unilateral or focal brain disease. Anisocoria is not uncommon in tabes, disseminated sclerosis, and paretic dementia.

Varying inequality of the pupils (*springing* or *alternating mydriasis*) or a one-sided mydriasis, now occurring on the one side and now on the other, is a serious premonitory symptom of insanity, and has been noted in general paralysis and locomotor ataxia. It is doubtful if it occurs in healthy persons, but the so-called false alternating mydriasis, according to Piltz and Frenkel, may be due to an inequality in the reflex excitability to light of the two eyes, or to inequality in the response to accommodation or spasm of the orbicularis muscles.

Special and Paradoxic Pupillary Phenomena.—The *hemiopic pupillary inaction* is referred to on page 518. Dilatation of the pupil under the influence of light stimulus and contraction when it has been shaded have been described in cases of meningitis as *paradoxic pupillary reactions.*

The phenomenon has been explained by assuming a reflex stimulation of the dilatator by a psychic influence, or that the action of the dilatator is indirectly increased because there is rapid exhaustion of the sphincter (Silex). The opposite condition, *paradoxic pupil dilatation*, is the antithesis of the condition just described, and has been observed frequently in experimental work in connection with the relation of the sympathetic to the eye. Paradoxic convergence reaction —that is, a dilatation of the pupil on convergence of the visual axes —has been recorded.

Hippus is a rhythmic contraction and dilatation of the pupil without

alteration of illumination or fixation. It is a normal phenomenon due to the constantly varying sensitive and psychic reflexes, but occurs in exaggerated degree in hysteria, mania, meningitis, and other diseases of the nervous mechanism.

Testing Acuteness of Vision.—The acuteness of vision is the power of distinguishing form and size, and is a function of the macula lutea, the peripheral portions of the retina having only indifferent ability to distinguish form and size.

In order to determine the acuteness of sight, test-types are employed, in which the letters are of various sizes, and constructed according to the methods described on page 35.

When it is desired to make the test, the patient is placed 6 meters from the type-card, in a well-lighted room, and each eye is tried separately. If the letters of No. 6 (20 feet approximately) are read, vision is normal, or 1, but if, at the same distance, letters no smaller than those numbered 18 (60 feet) can be discerned, vision is $\frac{1}{3}$. It is usual to express these results according to the formula $V = \frac{d}{D}$, in which V stands for visual acuteness, d for the distance of the patient from the card, and D for the distance at which the type should be read; so that in these instances the vision would be recorded $\frac{6}{6}$ and $\frac{6}{18}$, or in feet, $\frac{20}{\text{XX}}$ and $\frac{20}{\text{LX}}$. The rays coming from the letters at 6 meters' distance have so little divergence when they reach the eye that they are usually considered parallel. Hence, if the patient sees distinctly at this distance, his vision is perfect at the longest range (see also page 38). In point of fact, however, there is an appreciable divergence of the rays from the distance mentioned, equivalent to one-sixth of a diopter, and in the final adjustment of glasses this divergence should be recognized. Any other distance may be chosen, provided it does not place the patient closer to the test-card than 3 meters, at which close range the function of accommodation would introduce an element of inaccuracy. Thus, the scale made use of by de Wecker, and elaborated by Oliver, assumes $\frac{5}{5}$ $\left(\frac{15}{\text{XV}}\right.$ approximately$\left.\right)$ instead of $\frac{6}{6}$, as $\frac{1}{1}$. In like manner, a 4-meter distance may be utilized, as has been done by Edward Jackson. This author urges the standardizing of all cards of test letters by comparison of the visibility of each line of letters with that of a graded series of broken rings which he has designed and has had prepared.

The acuteness of sight, as tested with types constructed on the basis of an angle of 5′, does not always yield accurately the highest vision attainable; indeed, many good eyes possess a vision of $\frac{5}{4}$ of the standard angle. For this reason Dr. James Wallace has arranged a series of test-types in which an angle of 4′ has been substituted as the basis of each letter.

For the purpose of a control test, and also for determining visual acuteness of illiterate persons, cards are employed on which a number of differently arranged dots are placed, of sizes which should be counted

at different distances, and among these Burchardt's international tests are the most useful. For the same purpose incomplete squares corresponding in size to the test-letters have been constructed, the incomplete sides being turned successively in different directions. Wolffberg has designed a useful test which consists of small pictures of well-known objects, which in size approximately conform to the standard angle.

If the patient fails to decipher the largest letters at the distance employed, he should be moved closer to the card; thus, he may be unable to read the type numbered 60 at 6 meters, but may discern this at 4 meters, $V = \frac{4}{60}$ or $\frac{1}{15}$ of normal. Still further depreciation of visual acuteness is recorded by requiring the subject to count the outstretched fingers at various distances, 0.2, 1, or 2 meters, V = counting fingers at 0.2 meter, etc. For determining the lower degrees of sharpness of vision by a method more precise than the one just described, Landolt's *optotypes* may be employed.[1] If the patient is unable to count fingers, his ability to perceive the movements of the hand at 0.5, 1, or 2 meters is tested, V = movement of the hand at 0.5 meter, etc. When the ability to distinguish form (*qualitative light perception*) no longer exists, the perception of light should be investigated by alternately screening and shading the eye, by illuminating the eye with light reflected from a mirror, or focused upon it with a condensing lens.

Light-sense.—Having determined the acuteness of vision by means of the test-letters, the examiner has ascertained the *form-sense*, and may proceed to try a second subdivision of the sense of sight, the *light-sense*, which is the power possessed by the retina, or center of vision, of appreciating variations in the intensity of the source of illumination.

An instrument called a *photometer* is employed for this purpose, and consists essentially of an apparatus—for example, the one designed by Izard and Chibret—by which the intensity of two sources of light may be compared. The patient, looking into the instrument, sees two equally bright disks. One disk is now made darker, and the power of the eye to perceive the difference in the illumination of the two disks ascertained; or one disk is made entirely dark, and then gradually illuminated, and the smallest degree of light noted by which the patient can perceive the disk coming from the darkness. The former is called the *light-difference* (L. D.), and the latter the *light-minimum* (L. M.). In more exact language, to quote Percival Hay,[2] who has designed a photometer, by light-difference is meant the minimal difference capable of being perceived—the threshold of discrimination, and by light-minimum is meant the minimal stimulus capable of being perceived—the threshold of sensibility. By means of Förster's photometer the lowest limit of illumination with which an object is still visible (the *minimum stimulus*) is ascertained. The light-sense may also be tested with gray letters on a white ground, those of Bjerrum being constructed

[1] Ophthalmic Record, 1899, vol. viii, p. 624.
[2] Archives of Ophthalmology, 1905, vol. xxxiv, p. 160.

on the same principle as Snellen's types. For determining the "light-minimum" R. Wallace Henry's photometer is very useful. Ives and H. Maxwell Langdon have improved the accuracy of the last mentioned instrument by using electricity with a rheostat and ammeter in the circuit as the source of illumination, and having the amount of light controlled by an iris diaphragm instead of varying thicknesses of opal glass, as suggested by Henry. Some information in regard to the light-sense may be obtained by testing the acuteness of vision on two cards, under a different degree of illumination, and by comparing the results with a similar examination of a subject believed to have normal power of appreciating different degrees of illumination. De Wecker's *photometric types* may also be employed. These consist of white letters placed upon gray backgrounds of different intensities.

Color-sense.—A third subdivision of the sense of sight is the *color-sense*, or the power which the retina has of perceiving color, or that sensation which results from the impression of light-waves having a certain refrangibility. This examination is of especial interest in the detection of *color-blindness* (see page 496).

1. **Method of Holmgren.**—This consists in testing the power of a person to match various colors, conveniently used in the form of colored yarns. The set of worsteds contains three large test-skeins, namely: (1) *light pure green*, (2) *rose-purple*, (3) *red;* and 150 small skeins of the following colors: red, orange, yellow, yellow-green, pure green, blue-green, blue, violet, purple, pink, brown, and gray. In addition, there are several shades of each color, and a number of gradations of each tint, from the deepest to the lightest. According to Holmgren, the method of examination should be as follows:

"The wools are placed in a heap on a large table, covered by a light cloth and in broad daylight. A skein of the test-color is taken from the pile and laid far enough away from the others not to be confounded with them during the examination. The person examined is required to select other skeins from the pile nearly resembling it in color, and to place them by the side of the sample. He is made thoroughly to understand that he is required to search the heap for the skeins which make an impression on his chromatic sense, and quite independently of any name he may give the color similar to that made by the test-skein. The examiner should explain that resemblance in every respect is not necessary; that there are no two specimens exactly alike; that the only question is the resemblance of the color, and that, consequently, the candidate must endeavor to find something similar in shade, something lighter and darker of the same color, etc.

"Test I.—The green test-skein is presented. The examination must continue until the candidate has placed near the test-skein all the other skeins of the same color; or else, with these or separately, one or more of the skeins of the class of confusion colors, or until he has sufficiently proved, by his manner, that he can easily and unerringly distinguish the confusion colors, or gives unmistakable proof of a difficulty in accomplishing it. The candidate who places with the test-skein

confusion colors (gray, drab, fawn, light pink or yellow)—that is to say, finds that they resemble the test-color—is *color-blind;* while if he evinces a manifest disposition to do so, though he does not absolutely do so, he has a *feeble chromatic sense.*

"Test II.—The rose-purple skein is presented. The examination must continue until the candidate has placed all or the greater part of the skeins of the same shade near the sample; or else, simultaneously or separately, one or more skeins of the confusion colors. If he confuses the colors, he will select either the light or deep shades of blue and violet, especially the deep, or the light and deep shades of one kind of green, or gray inclining to blue. A candidate who is proved color-blind by the first test, and who in the second test selects only purple skeins, is *incompletely color-blind.* If in the second test he selects with the purple blue or violet, or one of them, he is *completely red-blind.* If in the second test he selects with purple only green or gray, or one of them, he is *completely green-blind.* The red-blind never select the colors taken by the green-blind, and *vice versâ.* The green-blind will often place a violet or blue skein by the side of the green, but it will then only be the brightest of these colors.

"Test III.—The red skein is presented. The test, which is applied to those completely color-blind, should be continued until the person examined has placed beside the test-skein all the skeins belonging to this hue, or the greater part, or else one or more confusion colors. The red-blind chooses besides the red, green, and shades of brown, which, to the normal sense, seem darker than red. On the other hand, the green-blind selects shades of these colors which appear lighter than red.

"The absence of all except one color sensation (*monochromatic vision*) will be recognized by confusion of every hue having the same intensity of light. *Violet-blindness* will be recognized by a genuine confuison of purple, red, and orange in the second test."

2. **Method of Thomson.**—The late Dr. William Thomson devised the following arrangement of the yarns: The set consists of a large green and a large rose test-skein, and 40 small skeins, each marked with a bangle having a concealed number, extending from 1 to 40, placed in a double box, so arranged as to keep the two series apart.

The large green skein being placed near by, the small skeins from 1 to 20 are placed in good daylight, and the employee under examination is directed to select 10 shades of the same color as the test-skein. One with normal vision will choose promptly the 10 greens with odd numbers.

A color-blind person will hesitate, and his selections will contain some even numbers, and the confusion colors will be shades of brown, etc., containing some red, or shades of gray, and will indicate the color defect. These figures are to be recorded on a blank, and the 20 skeins are to be removed. The large rose skein is then used, and the examination repeated in like manner with skeins numbered from 21 to 40, and the result recorded. The confusion skeins, which have even numbers, are blue, green, and gray. From the selections made by the man found color defective by the green test we are able to decide the character of

his color-blindness. Those selecting blues are red-blind, those taking greens and grays are green-blind, according to the nomenclature of Holmgren. There are 10 roses and 10 confusion colors in the second series.

3. **The Lantern Test.**—To control and also to substitute the various wool-tests, lanterns for detecting color-blindness are employed. Useful models have been designed by William Thomson, Charles H. Williams, and Edridge-Green. Concerning lantern tests, Dr. Thomson wrote as follows: "Whilst the wool-tests have been accepted universally as requisite for the detection of color defects, the employees of railroads and their friends have always objected to their use as having no relation to their daily duties, and have demanded such colors as are employed as signals. Furthermore, for two-fifths of the time during the night of an employee's life he is expected to govern his actions by colored lights, and hence a lantern which can imitate the night signals in form, color, intensity, and size, as they appear under all obstructions caused by rain, snow, fog, and smoke, is desirable. Its power over the wools to detect the central amblyopias of tobacco, alcohol, drugs, and disease, that would not be revealed by the skeins, makes it a necessity."

Dr. Thomson described his lantern as follows: "It consists of an asbestos chimney, which can be placed on the kerosene lamp in universal use on railroads, or over an Argand or other gas light, electric lamp, or spring candle-stick. Two disks, 4 inches in diameter, are so placed upon the chimney as to permit of their being superimposed partly. The lower disk contains seven glasses in apertures $\frac{1}{2}$ inch in diameter, having the white, red, green, and blue colors in general use on railroads. This may be considered the 'examination in chief,' whilst the upper disk, when combined with the lower by turning one or both, furnishes the 'cross-examination.' The upper disk has two apertures, one $\frac{1}{12}$ inch, the other $\frac{1}{2}$ inch, with white glass. The other five have one white ground glass, one deep London smoke, one pink, one green, and one cobalt-blue glass.

"The combination of the white ground and the smoke glass with the reds and greens of the lower disk enables all atmospheric conditions to be imitated, and the lights to be diminished in brightness and tint. The use of the small opening enables size and distance of signals to be imitated.

"The standard for color-sense is taken as an opening of $\frac{1}{12}$ inch at 20 feet. A man failing to see the colored light at this distance may have it increased ten times $= \frac{20}{200}$ by using the large openings. Again failing, he may approach to 1 foot and reveal a color-sense equal to $\frac{1}{200}$ only. The resemblance to the tests for form by Snellen's letter is to be noted. Since the color-blind depend alone on intensity of brightness to distinguish the white, green, and red signals, the diminishing effect of the ground glass and of the London smoke often reveals the defect. The cobalt, transmitting blue and red both, is usually described by the color-blind as blue, which color they always see well, being blind for red. The cobalt, combined with the lower reds, gives a very deep red color, which, when compared with the usual red, may induce the color-blind to name one red, the other green. Combined with the lower blue it gives a deep pink, called blue by the color-blind.

"In the pink, London smoke, and light green glasses in the upper disk I have imitated the 'confusion colors.' The pink looks cherry red to the normal eye, but it transmits both red and blue by the spectroscope, hence the color-blind pronounce it blue, or, when backed by a yellow flame, white. The light green is also called white, as is also the light gray of the London smoke. Hence we have in these three glasses tints which the color-blind name white, and reveal their defect thereby.

"The upper disk has its seven openings marked by the letters of the alphabet,

and the lower by the numerals from one to seven. The examination should be made in a darkened room, and the results reported on a blank, the details being used when requisite. The man examined is expected to call or name the colors and to recognize them at 20 feet."

4. **The Pseudo-isochromatic Plates of Stilling.**—These consist of a series of plates (10 in number), each plate containing 4 squares filled by small, irregular, colored spots, among which other spots in a confusion color, made to conform to an Arabic figure, are placed. The test-plate is held in a good light, and the examiner requires the subject to distinguish the tracings. These plates are said to be of practical use.

5. **Special Tests.**—These include the use of the *spectroscope* and various forms of *chromatometers*, the chromatophotometer of Chibret being the instrument that is perhaps most valuable.

Direct vision for colors may be studied by placing the patient at a given distance from a chart or disk of graduated colors, and noting the amount of surface exposure which is required for the color to be properly recognized. In the scale of De Wecker and Masselon the colored surface, 2 cm. square, should be recognized at 5 meters; that is, the chromatic vision or V. C. or C. = 1; if a colored test must be four times this size in order to be recognized, C = $\frac{1}{4}$, etc. Snydacker affixes to the ordinary type card squares of red (2.5 and 5 mm. in size) and green (3 and 6 mm. in size), the test being made at 6 meters. Failure to differentiate the larger test objects suggests the need of perimetric examination; also central scotomas for red and green may be thus rapidly detected (see page 85).

Accommodation has been defined to be those changes in the optical adjustment of the eye effected by the ciliary muscle, and in practice is measured by finding the nearest point at which fine print can be clearly deciphered, or by Duane's test (see page 38). The type usually adopted is that known as Snellen's 0.5 or Jäger's 1 (see also page 38).

In order to study the phenomena of accommodation the student should record: (1) The nearest point of perfectly distinct vision attainable with the smallest readable type, or the *punctum proximum* (abbreviated *p. p.*, or simply *p.*). (2) The farthest point of distinct vision, or *punctum remotum* (abbreviated *p. r.*, or simply *r.*). (3) The *range, amplitude* of accommodation, or the expression of the amount of accommodative effort of which the eye is capable. This is expressed in the number of that convex lens, placed close to the cornea, whose focal length equals the distance from the near point to the cornea, and which gives rays a direction as if they had come from the far point; thus, if the near point be 7 cm., the lens which expresses the amplitude of accommodation is $+ 14\ \mathrm{D}\ \frac{100}{7} = 14$. (4) *Relative accommodation*, or that independent portion of this function which can be exercised without alteration in a given amount of convergence, and which is divided into a *negative* portion, or that portion which is already in use, and

a *positive* portion, or that portion which is not in use (see also pages 35 and 46).

Mobility of the Eyes.—This is tested by causing the patient to follow, with his eyes, the head remaining stationary, the movements of the uplifted finger, which is directed to the right, to the left, upward and downward; or, better, a small electric light, the reflections of which from the corneas can be noted. Both eyes must be observed, and note made of any lagging in their movements, or of the failure of either eye readily to turn into the nasal or temporal canthus. At the same time the relation of the movements of the upper lid to those of the eyeball is recorded. The attention of the patient must be centered upon the moving test, and allowance should be made for the imperfect mobility of highly myopic eyes. Any asymmetry of the skull or difference in the level of the two orbital margins may be observed, because such conditions are not infrequently associated with ametropic eyes, especially when the two eyes possess great inequality in refractive conditions. (For accurate determination of ocular rotations see page 522, and Appendix, page 690.)

Balance of the External Eye Muscles.—Under normal conditions perfect equilibrium of the external eye muscles is present, and there is no interference with binocular fixation and binocular single vision (see page 521). Under abnormal conditions the movements of the eyes may be deranged so that one eye deviates, or tends to deviate, from the point of fixation—that is, from the object which it is regarding. These deviations may be classified thus:

1. *Manifest deviation*—that is, a deviation of an eye which the patient cannot overcome. This is known as *strabismus, squint,* or *heterotropia*, and is fully considered on pages 523 and 526.

2. *Latent deviation*—that is, a tendency of the visual line to deviate from the point of fixation. This tendency, however, is overcome by a muscular effort, owing to the stimulus which the eyes always have to maintain binocular single vision. It is generally described by the term *latent squint, suppressed squint,* or *heterophoria.* It is frequently designated *insufficiency of the ocular muscles,* and was called by von Graefe *dynamic strabismus* (see page 554).

According to G. T. Stevens,[1] the various conditions of equilibrium or variation from it may be arranged in four classes:

1. *Orthophoria,* a tending of the visual lines in parallelism.

2. *Heterophoria,* a tending of the visual lines in some other direction, but with ability to adjust them habitually for single vision.

3. *Heterotropia,* a deviation of the visual lines from parallelism in such manner that they cannot habitually be united at the same point of fixation.

4. *Anotropia, katotropia;* or *anophoria, katophoria*—variations from equilibrium which may or may not be consistent with parallelism of the visual lines, but in which, with the least innervation of the eye muscles, the visual lines of both eyes would fall below (katotropia) or rise above (anotropia) the most favorable plane for the minimum effort. Thus with ano- or katotropia there may be associated heterophoria or heterotropia.

[1] A System of Diseases of the Eye, edited by Norris and Oliver, vol. ii, p. 171.

Heterophoria may be divided into the following specific conditions:

1. *Esophoria,* a tending of the visual lines inward.

2. *Exophoria,* a tending of the visual lines outward.

3. *Hyperphoria* (right or left), a tending of the visual line of one eye (right or left) in a direction above its fellow, constituting, as the case may be, right or left hyperphoria.

The term does not imply that the line to which it is referred is too high, but that it tends higher than the other, without indicating which may be at fault.

The compound tendencies are:

1. *Hyperesophoria* (right or left), a tendency of one visual line to rise above the other, with a tendency also of the lines inward.

2. *Hyperexophoria* (right or left), a tendency of one visual line to rise above the other, with a tendency also outward.

Heterotropia may be divided into two subclasses:

(*a*) Deviations consistent with a physiologic state of the muscles and nerves, as in the ordinary concomitant squint.

(*b*) Deviations resulting from pathologic conditions—as, for example, deviations from paralysis or from mechanical causes.

The specific divisions of the subclass (*a*) are:

1. *Esotropia,* a deviation of the visual lines inward.

2. *Exotropia,* a deviation of the visual lines outward.

3. *Hypertropia* (right or left), a deviation of one visual line above the other.

4. *Hyperesotropia* and *hyperexotropia* are the compound deviations.

Cyclophoria is a term introduced by Savage to describe want of equilibrium of the oblique muscles.

In order to ascertain the condition of the ocular muscles the following tests are employed:

1. **The Screen (Cover) and Parallax Tests.**—Require the patient to regard a small point of light upon a black background 5 or 6 meters distant, or a round black spot 1 inch in diameter in the center of a white card-board at a similar distance. Cover the left eye with a screen, making sure that the patient is fixing the test-object with his right eye. Pass the screen rapidly from the left to the right, and observe the movements of the eye which take place behind the cover. Outward deviation indicates exophoria; inward deviation, esophoria; vertical deviation, hyperphoria. The prism, placed base inward, which neutralizes the outward deviation is a measure of the exophoria; the prism, placed base outward, which neutralizes the inward deviation is a measure of the esophoria; the prism, placed base up or base down, which neutralizes the vertical deviation is a measure of the hyperphoria.

While the screen is being moved rapidly from one eye to the other request the patient to describe the apparent movement of the test-object. If this moves in the same direction in which the cover is moved, exophoria is indicated; if in the opposite direction, esophoria; if upward or downward, hyperphoria. These apparent movements may be neutralized as before with appropriately placed prisms.

Require the patient to fix upon a fine object, as a pencil-point, held below the horizontal, 20 or 25 cm. from the eye, and, in order to remove the control of binocular vision, cover one eye with a card or the hand, and observe whether the eye under cover deviates inward or outward, upward or downward, and returns to fixation when the cover is removed, and neutralize the movement with appropriately placed prisms.

2. **Prism Tests.**—A small flame is placed against a dark background at 5 or 6 meters from the patient, and on a level with his eyes. In an accurately adjusted trial-frame a prism of 7° is inserted, base down, before one eye—for example, the right. Vertical diplopia is induced, and the upper image belongs to the right eye. If the flames stand one directly over the other, there is no inclination to divergence or convergence. If the upper image stands to the left, there is exophoria; if to the right, esophoria. That prism placed with its base in or out before the left eye, according to circumstances, which brings the two images into a vertical line, measures the degree of the deviation.

In order to test the functions of the vertical muscles at a distance of 6 meters the patient is seated as before, and a prism of sufficient strength to induce homonymous diplopia is placed before one eye,—for example, the right,—*i. e.*, with its base toward the nose. If the images are on the same level, no deviating tendency is present. If, for example, the right image rises higher than the other, the visual line of the right eye tends to be lower than that of its fellow, and there is hyperphoria. That prism, placed with its base down before the left eye, which restores the images to the horizontal level, measures the degree of deviation.

3. **Equilibrium Test.**—In order to test the functions of the lateral muscles at the ordinary working distance, or 30 cm., it is customary to employ the equilibrium test of von Graefe, in which a card, having upon it a large dot, through which a fine line is drawn, is held 25 or 30 cm. from the eyes, diplopia being induced by means of a prism of 10°, base up or down, before one eye. A more accurate test-object is a small dot and fine line, or a single word printed in fine type, requiring accurate fixation and a sustained effort of accommodation. If, the prism being placed base down before the right eye, the images stand exactly one above the other, equilibrium is evident; if the upper image (image of the right eye) stands to the left of the lower image, there is *crossed lateral deviation;* and that prism, placed before the left eye with its base toward the nose, which restores the images to a vertical line, measures the tendency to divergence or exophoria. If the upper image stands to the right of the lower, there is *homonymous lateral deviation;* and that prism, placed before the left eye with its base toward the temple, which restores the images to a vertical line, measures the tendency to convergence or esophoria. The vertical muscles should also be tested at the ordinary working distance with a prism placed before one eye, with the base before the nose, as already described above.

Edward Jackson's test for muscle balance at the working distance is useful. It consists of a small white square on a black ground, which is held at a distance of 33 cm. from the eyes, and which is regarded through a strong convex cylinder (10 or 12 D) placed before one eye. The white spot appears as a gray streak, which seems to pass through the spot if there is orthophoria, but to one or other side of it, or above or below it, if there is heterophoria. The axis of the cylinder must be vertical to test the vertical balance, and horizontal to test the lateral

balance. The Maddox rod test (see page 77) has also been adapted to the working distance, the test-object being a small illuminated square on a black ground, which is viewed at the near point through a Maddox rod. Schild and B. F. Baer, Jr. have designed useful instruments.

4. In order to test the **convergence near point,** approach a finger or pencil to the nearest point upon which the eyes can converge. This should be situated at no greater distance than 8 cm. ($3\frac{1}{2}$ inches) from the eyes—that is, 2.5 to 4.5 cm. ($1–1\frac{3}{4}$ inches) from the nose. If, before this point is reached, outward deviation of one eye occurs, the amount of convergence is deficient.

5. In order to ascertain the power of **adduction** (properly *prism-convergence*), **abduction** (properly *prism-divergence*), and **sursumduction** (*sursumvergence*), the strongest prism which the lateral and vertical muscles can overcome is found (see page 521).[1]

Beginning with *adduction* (*prism-convergence*), find the strongest prism placed before one eye, with its base toward the temple, through which the flame still remains single. The test should begin with a weak prism, the strength of which is gradually increased until the limit is ascertained. This varies from 30° to 60°, the higher degrees, however, in most instances being attained only after a reasonable degree of practice.

Fig. 32.—Risley's rotary prism.

In like manner *abduction* (*prism-divergence*) is tested, the prism now being turned with its base toward the nose; 6° to 8° of prism should be overcome. The ratio between adduction and abduction should be 6 to 1 (Stevens)—*i. e.,* if adduction is 48°, abduction should be 8°; but, according to Risley, in carefully corrected or emmetropic eyes, the ratio is 3 to 1. Banister found the primary adduction for 6 meters to be only 14°. As Hansell and Reber properly observe, no arbitrary standard of the ratio between prism-convergence and prism-divergence can be given, although the latter is fairly constant under normal conditions.

Sursumduction (*sursumvergence*), or the power of uniting the image of the candle-flame, seen through a prism placed with its base downward before one eye, with the image of the same object as seen by the other eye, is ascertained by beginning the trial with a weak prism, $\frac{1}{2}$° or 1°, and gradually increasing its strength. The limit is usually 2°, but may be as high as 6° or 8°. *Right sursumvergence* is equivalent to the degree of that prism placed base down before the right eye (or base up before the left), and *left sursumvergence* to the degree of that prism placed

[1] According to Duane, the term "adduction" is properly applied only to the amount (40°–50°) by which each eye can turn inward when moving parallel with its fellow (*associated adduction*, or adduction proper); "abduction," to the absolute degree of rotation of each eye outward in performing associated parallel movements, which is 40°–50°; "sursumduction," to the absolute degree of movement of either eye upward—a movement of some 40° in extent.

base down before the left eye (base up before the right) through which the test-object still remains single. Right and left sursumvergence are normally equal.

If the eyes of the patient under examination are ametropic, the proper correcting lenses should be placed before them, and the examination for the various forms of heterophoria made through this glass, which should be accurately centered.

Practically all the examinations for muscular errors can be made with a series of prisms and a trial-frame, but they are facilitated by the use of certain instruments of precision, especially some form of Herschel or *revolving prism*, the one devised by Risley being the best. The latter consists of two prisms, superimposed with their bases in opposite directions, constituting a total value of 45°. They are mounted in a cell which has a delicately milled edge, and fits in the ordinary trial-frame. The milled edge permits convenient turning in the frame, so that the base or apex of the prisms can be readily placed in any desired direction. The prisms are caused to rotate in opposite directions by means of a milled screw-head, projecting from the front of the cell.

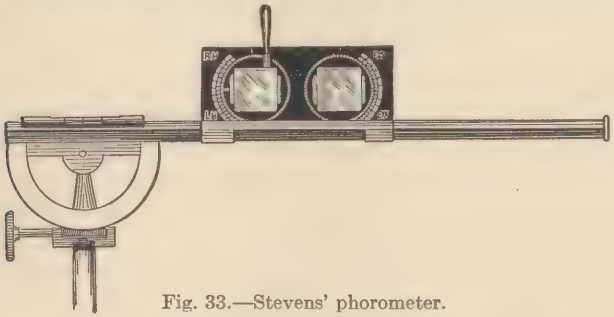


Fig. 33.—Stevens' phorometer.

With this rotary prism the strength of the abducting, adducting, and supra- and infraducting muscles can be measured. If the rotary prism is placed before the left eye with the zero mark vertical and the screw turned to the right or left, it will cause the base of the resulting prisms to be either inward or outward—that is, toward the nose or temple, as may be desired; or it may be placed with the zero mark horizontal, and the base turned upward or downward. All examinations for muscular defects may be ascertained with a *phorometer*. A number of excellent models are available.

6. **Obtuse-angled Prism Test.**—One of the simplest tests of the ocular muscles is the *obtuse-angled prism* of Maddox. This is composed of "two weak prisms of 3° united by their bases. On looking through the line thus formed at a distant flame, two false images of it are seen, one higher and one lower than the real image seen by the other eye, the position of which, to the right or the left of the line between the false images, indicates the equilibrium of the eye. A faint band of light, of the same breadth as the two false images, is seen extended between them" (Fig. 34). The answers of the patient may be materially assisted

by placing a red glass before one eye, and thus tinting the real image. If this stands directly in the center between the two false images, all forms of latent deviation are eliminated; if it stands to the right or to the left, there is exophoria or esophoria; if it stands above or below the center, or is fused with either the upper or the lower image, there is hyperphoria.

7. **Insufficiency of the oblique muscles** (*cyclophoria*), according to Savage, may be detected "by placing a Maddox prism, with its axis vertical, before one eye, the other being covered, which regards a horizontal line on a card 18 inches distant. This line appears to be two, each parallel with the other. The other eye is now uncovered, and a third line is seen between the other two, with which it should be parallel. Want of harmony in the oblique muscles is shown by want of parallelism of the middle with the other two lines, the right end of the middle line pointing toward the bottom and the left end toward the top line, or *vice versâ*, depending upon the nature of the case" (Fig. 35).

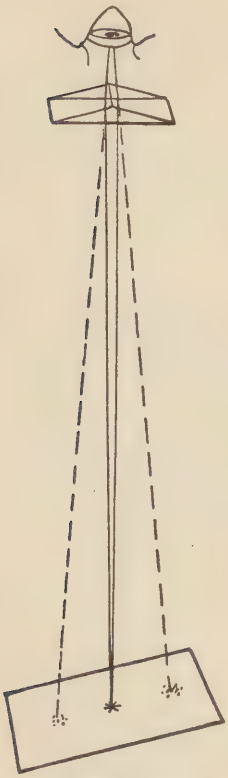


Fig. 34.—Position of the images as seen through the obtuse-angled prism of Maddox.

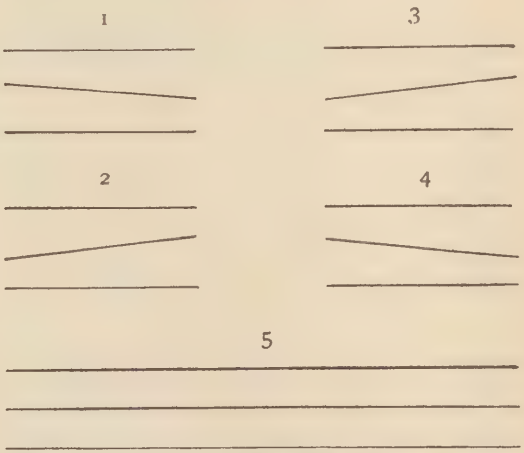


Fig. 35.—Tests for insufficiency of oblique muscles: 1, Insufficiency of left superior oblique; 2, insufficiency of left inferior oblique; 3, insufficiency of right superior oblique; 4, insufficiency of right inferior oblique; 5, equilibrium of oblique muscles (Savage).

8. **Cobalt Test.**—A trial-frame armed on one side—for example, the right—with a piece of *cobalt glass* is placed in position and the patient required to regard the test-light. The right image will be smaller than the left, and have a blue center and a red border if the patient is hyperopic or emmetropic, and a red center with a blue border if the patient is myopic. Suitably placed prisms, which unite the images, are the measures of the deviation.

9. **The Rod Test.**—This test was designed by Maddox, and depends upon the property of transparent cylinders to cause apparent elongation of an object viewed through them, so that a point of light becomes a line of light so dissimilar from the test-light that the images are not united. It may be suitably employed by having mounted in a cell, which will fit in the trial-frame, a transparent glass rod colored red, $\frac{3}{4}$ inch long, and about the thickness of the ordinary stirring rod used by chemists, or a series of glass rods placed one above the other (Fig. 36).

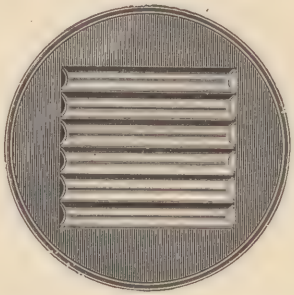


Fig. 36.—Maddox multiple rod.

The examination for *horizontal deviation* is thus described: "Seat the patient at 6 meters from a circle of light 5 mm. in diameter, and place the rod horizontally before one eye. If the line passes through the circle of light, there is orthophoria (equipoise), as far as the horizontal movements of the eyes are

Fig. 37.—Maddox's rod test for horizontal deviation. The rod is before the right eye. *a*, The line passes through the circle of light—orthophoria. *b*, The line passes to the right of the light—latent convergence, or esophoria. *c*, The line passes to the left of the light—latent divergence, or exophoria.

concerned. Should the line lie to either side of the circle of light, as in most people it will, there is either latent convergence or latent diver-

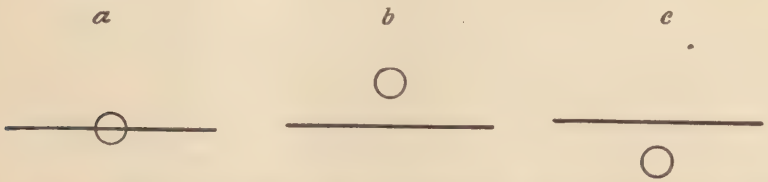


Fig. 38.—Maddox's rod test for vertical deviation. The rod is before the right eye. *a*, The line passes through the circle of light—orthophoria. *b*, The line passes below the light. The upper image belongs to the left eye—right hyperphoria. *c*, The line passes above the light. The upper image belongs to the right eye—left hyperphoria.

gence; the former, if the line is on the same side as the rod (homonymous diplopia); the latter, if on the other side (crossed diplopia)."

In order to test the *vertical deviation,* the rod is placed vertically before the eye; a horizontal line of light appears, and the patient is asked if the line passes directly through the flame or if it appears above or below it. The following rule, quoted from Maddox, will suffice to indicate the "hyperphoric" eye: "If the circle of light is lowest, there is a tendency to upward deviation of the naked eye; if the line is lowest, of the eye before which the rod is placed."[1]

The measurement of the extent of the deviation may be made in the ordinary way by finding that prism, placed before the naked eye (preferably with the rotary prism of Risley), which brings the line and flame together.

Of the various tests described, the Maddox rod is simple, and for all practical purposes accurate, especially when it is employed to estimate vertical deviations. According to Duane, it is apt to indicate an excess of deviation, particularly in esophoria. Hansell and Reber doubt if the prism-test reveals the true state of the muscle balance. They have found distinct contradictions between its results and those of the Maddox rod and other tests, and this is a matter of common experience. It is probable that the screen and parallax tests, if carefully and repeatedly performed, give, as Duane believes, most trustworthy information.

Power of Convergence.—In order to determine the maximum of convergence, an instrument known as an *ophthalmodynamometer* may be employed. The best one has been devised by Landolt,[2] and consists of a metallic cylinder, blackened on the outside, placed over a candle-flame. The cylinder contains a vertical slit, 0.3 mm. wide, covered by ground glass. The luminous vertical line thus produced is the object of fixation. Beneath the cylinder is attached a tape-measure graduated on one side in centimeters, and on the other in the corresponding number of meter angles. The fixation object is gradually approached in the median line toward the patient, until that point where double vision occurs is reached, or the nearest point (*punctum proximum*) of convergence, and the distance in centimeters read from one side of the tape, and the corresponding maximum of convergence in meter angles on the other.

The minimum of convergence may also be ascertained with the instrument, but when this is *negative* it is determined by finding the strongest abducting prism—that is, base in before one eye—which will not cause diplopia while the patient is fixing a candle-flame at 6 meters. If the number of the prism is divided by 7, the quotient will approximately give in meter angles the amount of deviation of each eye when the prism is placed before one. The amplitude of convergence is equivalent to the difference between the maximum and minimum of convergence —that is, $a = p - r$. Thus, if the normal average of maximum convergence is 9.5 meter angles and the average minimum of convergence is − 1 meter angle, the amplitude of convergence

[1] Dr. Swan M. Burnett substituted for the Maddox rod a 6 D cylinder.
[2] Landolt's Refraction and Accommodation of the Eye, p. 283.

would be $a = 9.5 - (-1) = 10.5$ meter angles. (See Meter Angles, page 44.)

The Field of Vision.—When the visual axis of one eye is directed to a stationary point, not only is the object thus "fixed" visible, but also all other objects contained within a given space, which is large or small, in proportion to the distance of the fixation point from the eye. This space is the *field of vision*, and the objects within it imprint their images upon the peripheral portions of the retina, or those which are independent of the macula lutea. In contradistinction to visual acuteness and refraction, which pertain to the macula in the act of *direct vision*, the function of sight capable of being performed by the rest of the retina is called *indirect vision*.

The limits of the visual field may be roughly ascertained in the following manner: Place the patient with his back to the source of light, and have him fix the eye under examination, the other being covered, upon the center of the face of the observer or upon the eye of the observer which is directly opposite his own, at a distance of 2 feet. Then let the surgeon move his fingers in various directions midway between himself and the patient, on a plane with his own face, until the limits of indirect vision are determined, controlling at the same time the extent and direction of the movements by his own field of vision. This method suffices to discover any considerable limitation, and, in the event of such discovery, should be supplemented by a more exact procedure.

If it is desired to have a map of the field not larger than 45° in extent, let the patient be placed 25 cm. from a blackboard, which may be conveniently ruled in squares, and fix the eye under observation upon a small white mark. The observer then moves the test-object—a piece of white paper 1 cm. square, affixed to a black handle—from the periphery toward fixation, until the object is seen. If eight peripheral points are marked and afterward joined by a line, a fair map of the field of vision will be obtained,[1] which may be transcribed upon a chart, like the one originally suggested by Joy Jeffries (Fig. 39). In like manner the *campimeter* of de Wecker may be employed. A useful hand campimeter has been designed by L. C. Peter.

Beyond 45° this method ceases to be accurate, because on a flat surface the object is too far away from the eye; rays perpendicular to the visual line coming from a peripheral object would be parallel to the

[1] The value in degrees of the squares on the blackboard may be ascertained by the following table, provided the eye is placed exactly at 25 cm. from the fixation point:

2.2	cm.	=	5°	in the perimeter semicircle.	
4.4	"	=	10°	"	"
6.7	"	=	15°	"	"
9.1	"	=	20°	"	"
11.7	"	=	25°	"	"
14.4	"	=	30°	"	"
17.5	"	=	35°	"	"
21	"	=	40°	"	"
25	"	=	45°	"	"
30	"	=	50°	"	"
36.7	"	=	55°	"	"
43.3	"	=	60°	"	"

blackboard, and could not arise from it, or any object passed across its surface.

Hence, the investigation of the periphery of the retina requires the use of an instrument known as a *perimeter*. This consists essentially of an arc marked in degrees, which rotates around a central pivot, that at the same time may be the fixing-point of the patient's eye, which is placed 30 cm. distant (the center of curvature of the perimeter arc), or the eye may be directed upon a porcelain button on a bar, placed 15° from the center, to the left, if the right eye is to be examined; *vice versâ*, if the left is under observation. The test-object, 1 to 2 cm. in diameter, affixed upon a carrier, is moved from without inward, and the point noted on each meridian where it is recognized. The result is transcribed upon a chart, prepared by having ruled upon it radial lines to

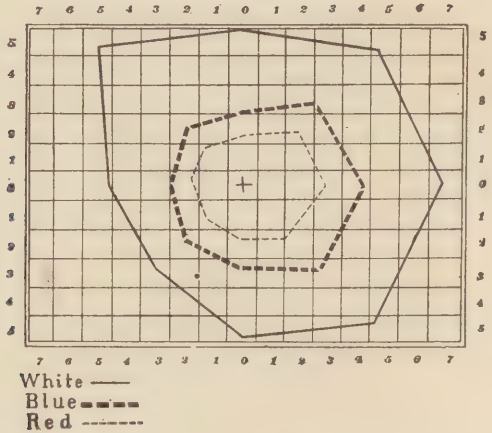


Fig. 39.—Limits of the normal field for white, blue, and red, transcribed upon a blackboard (after Norris).

correspond to the various positions of the arc, and concentric circles to note the degrees.

Many ingenious instruments have been devised, especially such as are self-registering, among which may be mentioned those of McHardy, Stevens, Skeel, and Priestley Smith. "Electric light perimeters" have been constructed by C. H. Williams and William Sweet. Rapid and accurate work, however, cannot be accomplished as satisfactorily with self-registering perimeters and a sliding object holder as with test-objects attached to a slender black rod or a slender wire. The hand perimeter of Schweigger for bedside examinations and the more elaborate one of Wildbrand, which can be adjusted while the patient lies upon his back, are useful instruments.

The physiologic limits of the form field, or, what is practically the same thing, the field when this has been mapped with a square of white 1.5 cm. in width, are: outward, 90°; outward and upward, 70°; upward,

50 ; upward and inward, 55°; inward, 60°; inward and downward, 55°; downward, 72°; downward and outward, 85°.[1]

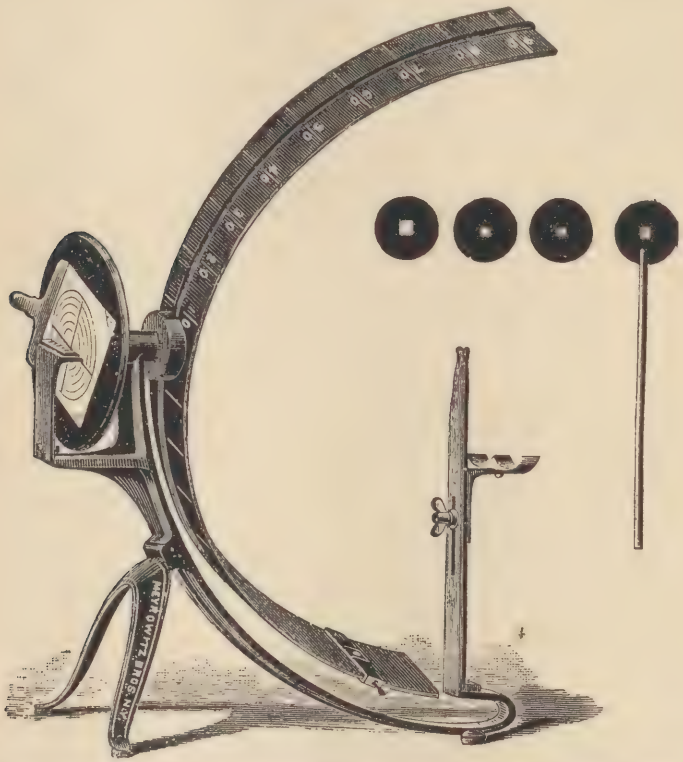


Fig. 40.—Perimeter. The examination may be made with the carrier which moves along the semicircle, or the test-objects may be carried along this by means of dark disks attached to a long handle, each disk containing in its center the test-object. The patient's chin is placed in the curved chin-rest; the notched end of the upright bar is brought in contact with the face, directly beneath the eye to be examined, which attentively fixes the center of the semicircle. The other eye should be covered, preferably with a neatly adjusted bandage. The record chart is inserted at the back of the instrument, and, by means of an ivory vernier, the examiner is enabled to mark exactly with a pencil the point on the chart corresponding to the position on the semicircle, at which the patient sees the test-object. The various marks are then joined by a continuous line, and a map of the field is obtained (see Fig. 41).

[1] Baas finds the average result of ten observers as follows: Outward, 99°; upward, 65°; inward, 63°; downward, 76°. Figures indicating a *minimal field*, or *smallest physiologic field*, have been recorded, varying from 90° (Foerster) to 50° (Treitel) outward; 55° to 21° upward; 60° to 40° inward; 70° to 40° downward. The smaller of these limits cannot be regarded as physiologic, and the greater is about equal to the average working field. Wolffberg insists that a field obtained under ordinary daylight illumination should be controlled by one obtained with reduced illumination. He further maintains that a field found to be "normal" in good daylight must, under reduced illumination, so long as the fixation spot remains visible, suffer no change in its outer limits and normal continuity.

Bjerrum, in 1899, proposed an addition to the usual method of examining the field of vision, a description of which is condensed from Berry's translation of the original paper. The addition consists in making use of white objects which subtend a very small visual angle. The examinations are made at a distance of 2 meters, using a large black screen 2 meters in breadth, which can be let down from the ceiling to the floor. At this distance the blind-spot (see page 85), instead of measuring 2.5 cm., as on an ordinary perimeter, measures 20 cm. in diameter; and everything else is in the same proportion.

The objects used by Bjerrum are small circular disks of ivory, fixed on the end of a

These measurements, which represent the *relative visual field*, vary within normal limits, and, transcribed upon a chart, produce the following figure (Fig. 41).

From this it is evident that the field of vision is not circular, being greatest in extent outward and below, and most restricted inward and above. This restriction is partly due to the presence of the edge of the orbit and the nose, and partly, as Landolt has pointed out, because the outer part of the retina is less used than the inner, and its functions, therefore, are less developed. Hence, as each portion of the field corresponds to the opposite portion of the retina, the inner part is smaller than the outer.

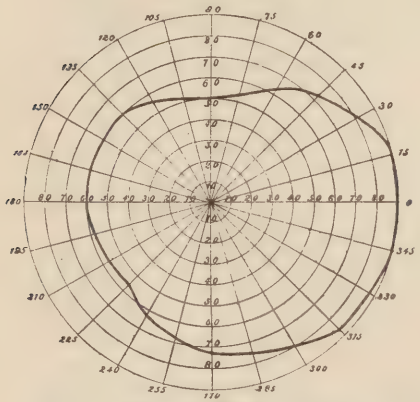


Fig. 41.—Diagram of the field of vision for white (1 cm. square test-object), transcribed upon a perimeter chart.

It must be remembered, however, that the size of the field of vision varies with the size of the visual angle subtended by the test-object with which the examination is conducted, and for the sake of accuracy the size of the object should be given as well as its distance from the patient's eye. Moreover, the visual field should not be mapped with test-objects of one size only. As Rönne, quoted by Traquair, says: "An examination of the field of vision in which only one object is used is in itself just as inadequate as an examination of visual acuteness with a test card which has letters of only one size."

Binocular Field of Vision.—The field of vision for each eye having been defined, it remains to point out that the field of vision which pertains to the two eyes, or that portion in which binocular vision is possible, constitutes only the area where the central and inner parts

long, dull black rod. They vary from 10 to 1 mm. in diameter. The examination is begun in the ordinary manner (at 30 cm.), with the 10-mm. disk, and then continued at 2 meters' distance with a 3-mm. disk. In the first case the visual angle approximately is 2°; in the second, 5′. The normal boundaries in the first instance have been given; in the second they are 35° outward, 30° inward, 28° downward, and 25° upward. Small concentric limitations are unimportant, but the method is valuable in finding sector-shaped defects, irregular limitations, and scotomas, and should supplement ordinary perimetric work in all important examinations (see page 85). W. G. Sym and A. H. H. Sinclair have well described the necessary apparatus for Bjerrum's test for scotomas in the field of vision (Ophthalmic Review, 1906, vol. xxv, p. 141); T. B. Holloway has designed a conveniently mounted set of Bjerrum's tests, white and colored (Transactions of American Ophthalmological Society, 1911, vol. xii, p. 966); and H. M. Traquair (Ophthalmic Review, 1914, vol. xxxiii, p. 65) gives an excellent account of the *quantitative method* in perimetry. Clifford B. Walker, in his examinations of patients with intracranial lesions in Harvey Cushing's service, finds it necessary to investigate the field for test-objects varying in visual angle from 1 or 2 minutes to about 8°, and has designed a series of nine circular rimless colored and white disks supported on wire handles which cover the required range, taking the "normal" or 5-mm. disk as the unit. In this series the smallest disk is 0.15 mm. and the largest 4 cm. in diameter. His special perimeter has a radius of 28.6 cm. and a large working surface 60° by 130° in extent.

overlap. This is evident from the diagram. The continuous line *L* bounds the field of vision of the left eye, and the dotted line *R* the visual field of the right eye. The central white area corresponds to the portion common to both eyes, or to that area in which all objects are seen at the same time with both eyes; the shaded areas correspond to the portions in which binocular vision is wanting. In the middle of the white area lies the fixation point *f*, and on each side of it the blind-spots of the right and left eye, *r* and *l*.

Having thus determined the *limits* and *continuity* of the visual field, the functions of the peripheral parts of the retina in regard to perception of colors, acuteness of vision, and appreciation of light should be investigated.

The *color-field* is mapped in the manner described in connection with the general visual field, the squares of white being replaced by pieces of colored paper.

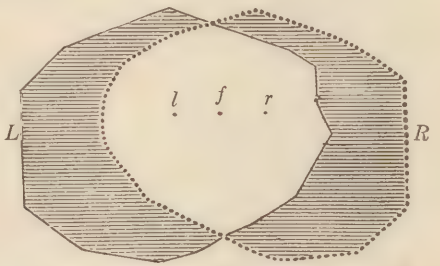


Fig. 42.—Binocular field of vision (Moser).

The order in which the colors are recognized from without inward is: (1) Blue; (2) yellow; (3) orange; (4) red; (5) green; (6) violet. In practical work, blue, red, and green are the colors employed, red and green being the color-sense most usually affected in pathologic cases. Non-saturated colors are not correctly recognized when the test-object is first seen. Thus, yellow at first appears white; orange, yellow; red, brown; green, white, gray, or gray-blue; and violet, blue. The physiologic limits of the color-fields, which, like those of the general field, are subject to variations, when estimated with a 1 cm. square test-object, the eye being 30 cm. from the fixing point, correspond closely to the following:

	Blue.	Red.	Green.
Outward	80	65	50
Outward and upward	60	45	40
Upward	40	33	27
Upward and inward	45	30	25
Inward	45	30	25
Inward and downward	50	35	27
Downward	58	45	30
Downward and outward	75	55	45

These, when transcribed upon a chart, are represented in Fig. 43.

The numbers represent the usual limits at which the color-test 1 cm. square is recognized as such. They do not indicate its greatest intensity,

which is perceived only at the fixation point. In order to avoid discrepancies, the character of the light, the nature and saturation of the color, and the size of the test-object should be stated in describing examinations. Always the visual field for colors as well as for white should be investigated with test-objects of various sizes.

It should be remembered that the boundaries of the color-field which have been described result from examination with test-objects not greater than 1 or $1\frac{1}{2}$ cm. square. With larger areas of color it will be found, as Gowers and others have shown, that the color-fields differ in extent very little from the fields for white.

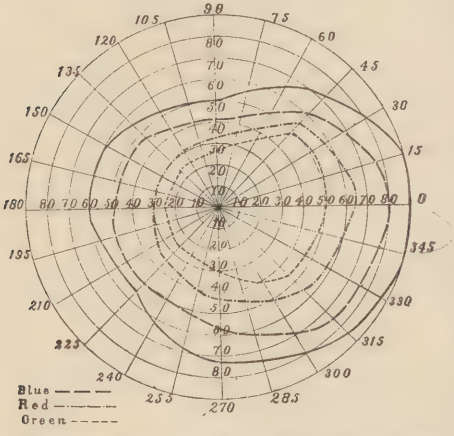


Fig. 43.—Diagram of the field of vision for blue, red, and green. The outer continuous line indicates the limit of the form-field; the broken lines, the limits of the color-fields.

According to Wolffberg, the color-limits contract concentrically as the illumination is reduced, but if the photochemical and neuroptic apparatus is normal, there will be no change in the normal sequence of the color-limits. Blue should be employed in investigating defects in the photochemical apparatus, as it is the color first to disappear in reduced illumination; red suffers promptly in reduced excitability of the neuroptic apparatus.

The *acuteness of the vision* of the *peripheral parts of the retina* may be tested with small squares of black paper (6, 5.3, and 2 mm. black quadrants on a white ground), separated from one another by their own width, by noting the point in each meridian where they are recognized as separate objects; or with gray patches of different intensity on a white ground (Ward Holden).

The *light-sense* of the *periphery of the retina* may be tested conveniently with Ward Holden's tests. One card has a 1 mm. black point on one side and a 15 mm. quadrant of light gray, having four-fifths of the intensity of white, on the other. With a perimeter of 30 cm. radius the black point and gray patch are each seen by a normal eye outward, 45°; upward, 30°; inward, 35°; downward, 35°. The second card has a 3 mm. black point on one side and a darker gray patch, having three-fifths of the intensity of white, on the other. Each is seen on the perimeter arc outward, 70°; upward, 45°; inward, 55°; downward, 55°. Card 2 will reveal slight disturbances of light-sense near the periphery and card 1 in the intermediate and central zones.

The *perception of light*, according to the experiments of Landolt, is the most constant function of the healthy retina, and remains nearly the same throughout its surface, while the color- and form-sense rapidly

lessen toward the periphery. For practical purposes, a candle-flame passed along the arm of the perimeter may be used as a test-object; and, if vision is very defective, a second candle is made the point of fixation. Progressive diminution of light-sense from center to periphery will be found if test-objects of varying luminous intensity, with the illumination of ordinary daylight, are employed.

The *adaptation of the retina* may be estimated according to Wilbrand's method by investigating the visual field in a dark room with test-object and fixation point streaked with luminous paint. The examination is made as soon as the patient enters the dark room and again in ten minutes. This interval is sufficient to enable the normal eye to adapt itself so that the extent of the visual field corresponds to that of a white object in diffuse daylight. Delayed adaptation is a phenomenon found in many pathologic conditions.

Abnormalities of Visual Field and Scotomas.—The most frequent departures from those limits of the visual field assumed to be normal are general or concentric contraction; contraction limited especially to one or the other side; peripheral defects in the form of re-entering angles; absence of one segment or quadrant; and absence of the entire right or left half of the field.

In addition to these defects, search should be made for dark areas within the limits of the visual field, or *scotomas*. These are distinguished as *positive* when they are perceived by the patient in his visual field, and *negative* when within the confines of a portion of the visual field the image of an external object is usually not perceived, and the affected area is generally not discovered until the field is examined. Negative scotomas, however, may also be positive in the sense that the affected areas of the retina which are insensitive to luminous impression can be recognized as dark areas and be projected exteriorly (Fuchs). Negative scotomas are further divided into *absolute* and *relative*. Within an absolute scotoma all perception of light is wanting, while within the confines of a relative scotoma the perception of light is merely diminished. The latter are *color scotomas*, usually for red and green. Scotomas are further subdivided, according to their situation and form, into *central*, *paracentral*, *ring*, *peripapillary*, and *peripheral*.[1] A normal eye will develop a relative central scotoma in a darkened room, and if the darkness is increased the scotoma becomes absolute (Hess).

In every normal eye there is a physiologic scotoma, corresponding to the position of the optic nerve entrance, which usually may be found 15° to the outer side of and 3° below the point of fixation; the interval, according to Landolt, being greater in hyperopic than in myopic eyes. This is known as *Mariotte's blind-spot*. It is surrounded by a narrow zone of relative amblyopia for white about 1° in width. According to Hansell, the average distance of the center of the blind-spot from

[1] For a scotoma which lies between the fixation point and the blind-spot, such as occurs in toxic amblyopia (see page 489), Traquair proposes the name *centrocecal scotoma*, and for other defects of the central part of the field the terms *supracentral*, *infracentral*, *nasocentral*, *temporocentral*, and for defects adjacent to the papilla, *supracecal*, *temporocecal*, etc.

fixation point is almost identical in emmetropia and hyperopia, but in myopia is about 5 mm. greater. Should the horizontal diameter of the blind-spot for motion exceed 6°, according to Van der Hoeve, it is larger than normal, and may indicate the presence of beginning pathologic change.

For the detection of scotomas, small test-objects, white or colored, $\frac{1}{4}$ cm. square, are employed, which are moved in different directions from the point which the eye under observation attentively fixes, and the spot marked where the object begins to disappear or change its color. The arm of the perimeter is usually marked near the center in half-degrees for this purpose. All examinations around the center of the field of vision, and hence the examination for scotomas, are readily made upon a blackboard or a campimeter.

Berry urges that the ordinary test for scotomas be supplemented by making an examination of the particular area of the field at a distance of 2 meters or more, so as to obtain a larger projection of the blind portion, and to be able to work with smaller retinal images, without necessitating the use of very small objects (see Bjerrum's method, footnote, page 81). Special instruments for detecting and measuring scotomas—*scotometers*—have been designed by Priestley Smith and P. C. Bardsley. Haitz employs a stereoscope with diagrams which gives binocular fixation. The card before the eye to be tested is covered with small squares with which the defect is detected and mapped; each side of a square subtends an angle of 1 degree at the distance at which the card is used.[1]

Tension.—This term indicates the resistance of the ocular tunics, and is clinically demonstrable by palpating the globe with the finger-tips. The middle and ring-fingers are placed upon the brow of the patient, the tips of the index-fingers upon the eyeball, and gentle to-and-fro pressure made, the eyes being directed downward. This pressure must be made in such a manner as not to push the ball into the orbit; otherwise no information of its true resistance is obtained. The tension of one eye must always be compared with that of its fellow, and, in any doubtful case, the results may be contrasted with those obtained by examining an eye known to be normal in another patient of similar age.

Normal tension is often expressed by the sign Tn, and the departures from it formerly were indicated by the symbols + ?, + 1, + 2, + 3, and − ?, − 1, − 2, − 3; the plus signs referred to increased, and the minus signs to decreased, resistance. In physiologic experiments, various kinds of apparatus, constructed upon the principle of the manometer, are employed, and for clinical purposes instruments known as *tonometers* have been devised. The most useful of these is the one designed by Schiötz. A modification of this tonometer has been constructed by Gradle, and W. McLean has designed a direct reading tonometer with constant weight factor.

[1] For a full description of various instruments designed for detecting and measuring scotomas, see J. Jameson Evans' article on "The Field of Vision" in The Ophthalmoscope, 1911, vol. ix, p. 699.

The principle of the eye tonometer is thus defined by Priestley Smith: The instrument measures the impressibility of the eyeball, and from the degree of impressibility we infer the intra-ocular pressure.

The Schiötz instrument is now in common use, and its construction may be readily understood from the accompanying diagram (Fig. 44).

The patient should lie upon a table or reclining chair, with the head thrown back, and be requested to look directly upward. The eyeball is rendered insensitive with holocain (2 per cent. solution, three instillations at intervals of three minutes; Marple prefers a 2 per cent. solution of cocain) and the lids are separated without making pressure on the globe. Next, the surgeon, holding the instrument (with the 5.5 gm. weight in place) by the two arms attached to the cuff, which slips up and down the hollow cylinder, brings its foot-piece exactly at rest on the cornea, the cuff being slid down so as to be about the middle of the cylinder. The instrument should stand freely, resting exactly on the center of the cornea. The deflection of the needle is recorded, and from a diagram which accompanies the instrument it may be seen with how many millimeters of mercury the deflection of the needle corresponds; thus, should the needle, with the 5.5 gm. weight in place, register 3, this corresponds to 25 mm. of mercury. Schiötz advises that three measurements shall be made and the average of the readings recorded. Some surgeons prefer a single reading taken with great care, but, as Priestley Smith insists, it is not safe to rely on a single reading; it should be confirmed by at least two additional readings. There are four weights, respectively 5.5, 7.5, 10, and 15 gm. If the needle is not deflected beyond 1, the measurement should be repeated with a greater weight. According to Schiötz, deflections between 2 and 4 mm. yield the most accurate results. The foot-piece of the instrument should be cleansed in a boric acid solution.

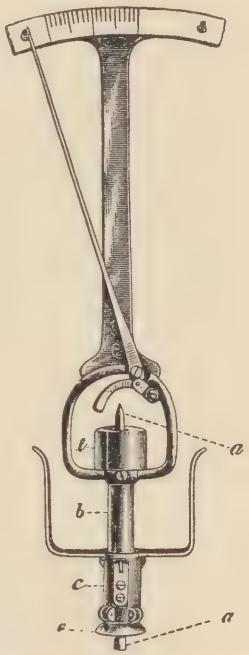


Fig. 44.—Schiötz tonometer.

The normal pressure, it is usually stated, varies from 14 to 28 mm. of Hg., that is, 14 is the low and 28 the high physiologic limit, although an eye with a tension above 25 should be regarded with suspicion. According to Schiötz, normal tension varies between 15.5 and 25; according to Marple, between 15 and 25; according to Stock, between 12 and 26, and according to Heilbrun, between 12 and 27. Neither the state of the refraction nor the age has an appreciable effect on the ocular tension, nor is it influenced by mydriatics, except that cocain slightly diminishes the tension of the normal eye (Heilbrun). According to Heilbrun, eserin and pilocarpin usually lower slightly the tension of the normal eye, an observation which is not confirmed by Marple's results. In this respect the author's observations agree with those of Heilbrun. Although records in the form of mm. Hg., as stated above, are usually made, as Priestley Smith points out, the reading and not the supposed equivalent in mm. Hg. should be stated (see also page 368). To

estimate the ocular tension by means of the finger test is not a safe nor an accurate procedure, and in all cases where this is an important observation the tonometer should be used.

Proptosis, exophthalmos, or protrusion of the eye, may be caused by orbital diseases and tumors, sinusitis, tenotomy and paralysis of the ocular muscles, Graves' disease, and sometimes by chronic nephritis (L. Barker), and by intracranial neoplasms, especially if situated in the middle fossa of the skull; while enlargement of the ball is the result of various conditions residing within the globe—myopia, buphthalmos, intra-ocular tumor, and staphyloma. If the cause is unilateral, the resulting condition is asymmetric, and the two eyes may be compared by observing the relative positions of the apices of the corneæ with each other and with the line of the brows. For measuring the degree of exophthalmos Edward Jackson has devised a simple scale or *proptometer*. A useful and accurate instrument for this purpose is the *exophthalmometer* of Hertel. A more elaborate instrument is the one designed by Lohmann. A slight protrusion of the eyeball takes place when the palpebral opening is voluntarily decidedly widened (see also page 592).

The eyeball is apparently sunken (*enophthalmos*) in some cases of ptosis, in wasting of the orbital fat, in orbital injury, and is diminished in size in high grades of hyperopia and congenital failures of development. As Nettleship has pointed out, the amount of exposed sclera decides the apparent protrusion or recession of the eyeball (see also page 592).

Position of the Eyes.—Instead of presenting parallel visual axes, one eye may be deviated inward, outward, downward, or upward, constituting one of the various types of strabismus (see page 523), a condition which may or may not be associated with diplopia (see page 528).

CHAPTER III

REFLECTION, THE OPHTHALMOSCOPE AND ITS THEORY. OPHTHALMOSCOPY AND SKIASCOPY

Reflection.—When light falls upon a polished surface a portion of it is reflected. The angle of reflection is always equal to the angle of incidence. A polished surface, capable of reflecting light, is called a *mirror*. Mirrors are *plane, concave,* or *convex.*

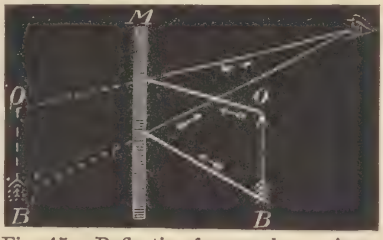


Fig. 45.—Reflection from a plane mirror.

A plane mirror reflects the rays falling upon it, so that they seem to come from a point as far back of the mirror as the object lies in front of it. It does not render the rays either convergent or divergent, nor does it lessen their convergence or divergence. Rays parallel before reflection are parallel after reflection. Rays convergent or divergent before reflection maintain the same relation after reflection. In the figure, rays from the object *O–B*, falling upon the mirror, *M*, are reflected so that they enter the observer's eye, and seem to him to come from *O′–B′*, situated as far back of the mirror as *O–B* is in front of it. The image is

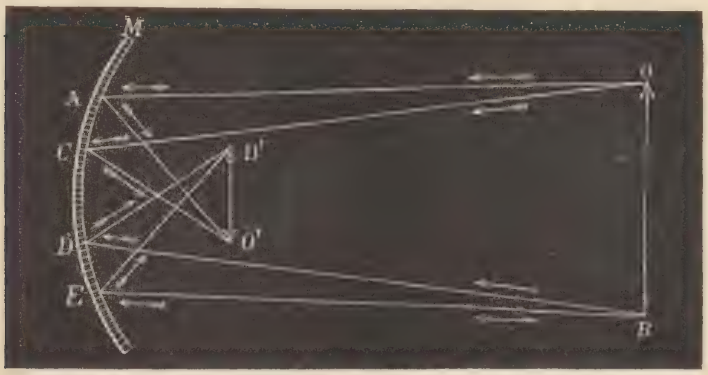


Fig. 46.—Reflection from a concave mirror.

not inverted. The rays have a divergence from a point whose distance is equal to the sum of the distance from the light to the mirror, and of the distance from the mirror to the eye (compare Skiascopy).

A concave mirror converges parallel rays of light to its principal focus, and forms a real, inverted image in front of the mirror.

The principal focus of a concave mirror is equal to one-half the length of its radius of curvature, $F = \frac{r}{2}$.

The conjugate focal distance for any point greater than the principal focus may be found by the following formula: f' represents the distance from which the rays diverge (the lamp or candle); f'' is the distance of the conjugate focus.

$$\frac{1}{f'} + \frac{1}{f''} = \frac{1}{F}.$$

$$\frac{1}{f''} = \frac{1}{F} - \frac{1}{f'}.$$

This is understood by recollecting that F is the focus for parallel rays, and that the focus is the inverse of the reflective or catoptric power of the mirror. The rays which diverge from f' require $\frac{1}{f'}$ of catoptric power to render them parallel. This diminishes the catoptric power of the mirror to $\frac{1}{f''}$.

$\frac{1}{F} - \frac{1}{f'} = \frac{1}{f''}$, the focal length of f'' is the conjugate focal distance required.

Example.—The ophthalmoscopic mirror has a focus of 20 cm., its radius of curvature being 40 cm. A candle is situated at 30 cm. in front of it, and we wish to know the conjugate focal distance:

$$F = 20 \text{ cm. } f' = 30 \text{ cm. } \frac{1}{20} - \frac{1}{30} = \frac{1}{f''}, \frac{1}{f''} = \frac{1}{20} - \frac{1}{30} = \frac{1}{60}, f'' = 60 \text{ cm.}$$

The rays of the candle would be rendered convergent to a point 60 cm. in front of the mirror. The light being placed at a greater distance than the principal focus, the rays are convergent.

A convex mirror renders parallel rays divergent as if they came from its principal focus, which is *negative*, situated behind the mirror, at a distance equal to one-half the radius of curvature. The image is erect and small.

The conjugate focal distances for convex mirrors are obtained by the same formula as for concave mirrors, the sign — being prefixed to F and f''.

The cornea, by reflecting light, corresponds to a convex mirror, and in this relation is important in ophthalmometry. The principal focus of the corneal mirror is about 4 mm., the radius of curvature being 7.829 mm. The size of the image reflected from the cornea is proportional to the size of the object as the focus of the corneal mirror, 4 mm., is to the distance of the object. A candle-flame 20 mm. in diameter, situated at 100 mm., gives a corneal image whose size is found in this manner: Image : 20 : : 4 : 100. $\frac{Image}{20} = \frac{4}{100}$. Image = 0.8 mm.

If the radius of curvature is greater, the image is also greater; if the radius of curvature is smaller, the image is smaller. By this means curvature ametropia may be measured.

The size of the cornea image is so very small that it would not be feasible to attempt direct measurement of it. If two candles which are separated some distance are employed as an object, each candle represents one extremity of the object. The size of the object is, then, the distance between the two candles; the size of the image is the distance between the reflected images of the candles. Suppose this distance to be 3 mm. and by means of a double refracting prism two images of each candle are seen; if they are displaced by the prism exactly 3 mm., so that a straight line passes through all the images, two of them must overlie, as the images are 3 mm. apart. Small variations in curvature will now be manifest if the two images, which should overlie exactly, shoot past each other or fail to come together. The change of form in the crystalline lens during accommodation is proved by this experiment.

THE OPHTHALMOSCOPE

For the purpose of studying the interior of the living eye an instrument known as the *ophthalmoscope*, the invention of which, in 1851, we owe to the genius of von Helmholtz, must be employed. The original Helmholtz ophthalmoscope was composed, in general terms, of three thin glass plates, set in a suitable frame at an angle of 56 degrees to the line of sight, by means of which the light was reflected into the observed eye. With this instrument the details of the eye-ground can be studied under a weak illumination.

The modern ophthalmoscope consists essentially of a concave silvered mirror for illuminating the eye, and of lenses for measuring and modifying its refraction (*refraction ophthalmoscope*). The mirror is perforated, as originally suggested by Reute, and swings to either side, so that the obliquely incident rays may be reflected into the eye without having to tilt the entire instrument, and thus narrow the aperture and render the lenses astigmatic. The lenses are inserted in a disk, invented by Rekoss, which can be rotated in front of the sight-hole. A plane mirror, which can be substituted for the concave mirror, is a valuable addition. Many ophthalmoscopes contain two disks, which can be used either singly or in combination. This arrangement affords a series of lenses from 0.50 to 24 D concave, and from 0.50 to 23 D convex, with which the observer is enabled to view distinctly the details of the eye-ground in all forms of ametropia. A lens varying from 13 to 20 D accompanies the instrument for focal illumination of the cornea and lens, and for use in the indirect method of ophthalmoscopy. Among the many ophthalmoscopes at the student's disposal, in the author's opinion none is better than the Loring instrument (Fig. 47). Excellent models have been designed by Edward Jackson, B. A. Randall, and A. S. Morton.

Electric ophthalmoscopes, for which we are largely indebted to W. S. Dennett, are very useful in the examination of bedridden patients. The

source of illumination consists of an electric bulb in the end of the handle, the light of which is condensed by a lens on a suitably tilted mirror which reflects it into the eye; indeed, they are essential for accurate work. W. B. Marple's electric ophthalmoscope is, in the opinion of the author, the best of these instruments thus far designed (Fig. 48). A convenient model is the one designed by George Crampton, the handle of which contains the electric battery, and admirable ophthalmoscopes

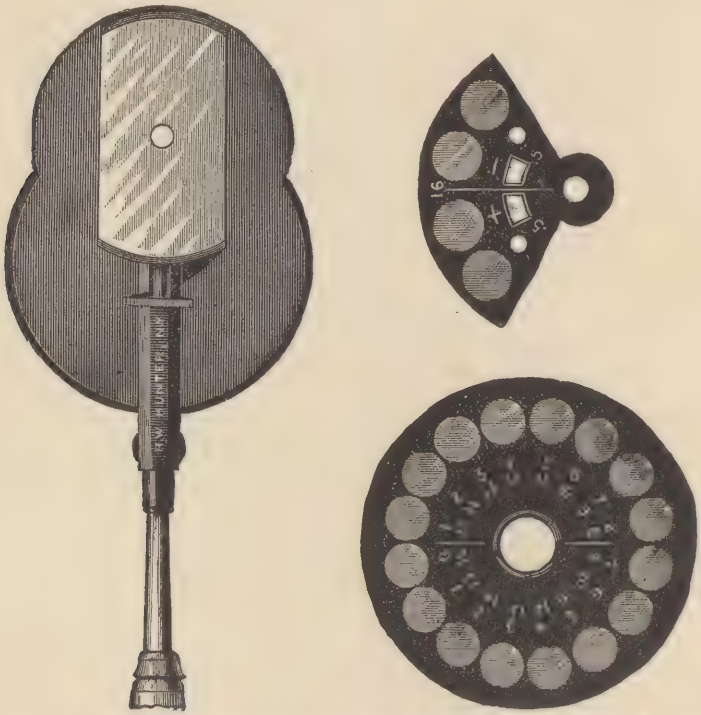


Fig. 47.—Loring's ophthalmoscope, with tilting mirror, complete disk of lenses from − 1 to − 8 and 0 to + 7, and supplemental quadrant containing ± 0.5 and ± 16 D. This affords 66 glasses or combinations from + 23 to − 24 D.

of this character have been devised by S. Lewis Ziegler, Claiborne, and Charles H. May.

Large demonstrating ophthalmoscopes have a distinct value in ophthalmoscopic work. The Gullstrand ophthalmoscope is the most perfect instrument of the type. It is free from annoying reflections and furnishes magnifications from 5 to 40 times in monocular and 20 times in binocular observations.

Direct Method.—The rays from the concave mirror, somewhat converging, enter the pupil and are brought to a focus in the vitreous humor. After reaching their focus the rays diverge again and spread out on the retina into a circle of diffusion. The portion of the retina thus illuminated sends rays back again, which pass through the dioptric

media of the eye and are refracted to its far point—that is, if the eye is emmetropic, they emerge parallel and would meet at an infinite distance; if the eye is myopic, they converge to their far point in front of the eye; if the eye is hyperopic, they diverge from their far point back of the eye (see also page 106).

An observer's eye, in order to focus these rays, must be adapted to them. If the patient is emmetropic, the observer's eye must also be rendered emmetropic. If the patient is hyperopic, the emmetropic

Fig. 48.—Marple's electric ophthalmoscope.

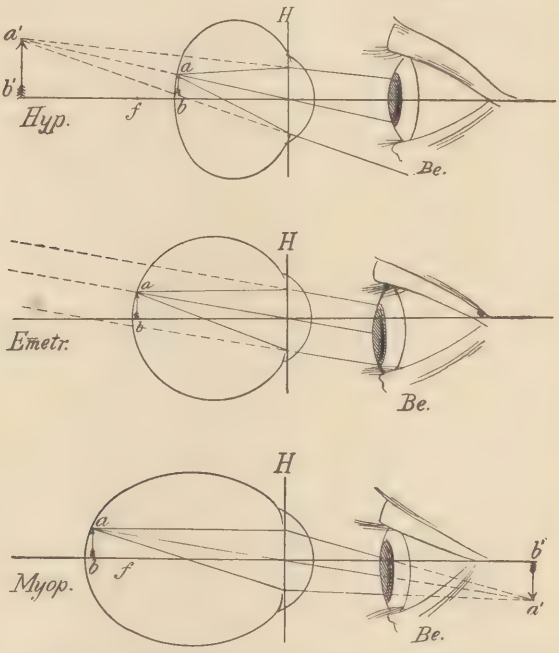


Fig. 49.—Examination in the erect image when the eye examined is hyperopic, emmetropic, or myopic. In each figure three rays are shown emanating from a luminous point on the eye-ground. In hyperopia they diverge after leaving the eye; in emmetropia they are parallel; in myopia they converge. *f*, The posterior focus; *H*, principal plane of the dioptric system of the examined eye; *Be*, observer. The ophthalmoscope is not shown (Haab).

observer must add a convex glass to his eye, or use his accommodation, in order to make the divergent rays parallel. If the patient's eye is myopic, the emmetropic observer must place a concave glass before his eye to render the convergent rays parallel. If the observer is ametropic, he must first correct his ametropia with suitable lenses (see also page 107). A hyperopic observer might see distinctly the eye of a myopic patient, or a myopic observer might see the eye of a hyperope. In either case the hyperopia must at least be as great as the myopia.

In this method the observer sees the eye just as he would see an object through a convex glass or simple microscope. The image of the eye-ground is a virtual one—that is, it seems to be behind the eye. It is magnified and erect.

The *formation of the image* in the *direct method* may be understood by examining Fig. 50.

Divergent rays and convergent rays have been described, but always in relation to one point (see page 22). It is now necessary to consider their meaning in reference to an image.

An image is composed of a succession of points; each one of these points represents a point in the object. From the point in the object one ray passes to the optical center of the lens or lenses, and maintains

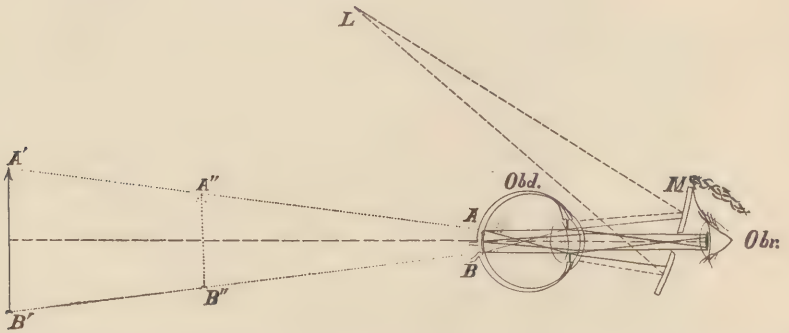


Fig. 50.—Diagram of the direct method with the formation of an upright image: Rays from the source of light, *L*, are received upon the concave mirror, *M*, and converged upon the observed eye, *Obd*, within which they cross and illuminate an area of its fundus. From an area *A*–*B* thus lighted, rays pass out of the pupil (parallel if it be emmetropic, as here represented) through the sight-hole of the mirror, and, entering the observer's eye, *Obr*, are focused upon his retina. An image is there formed as though the object seen were at a great distance, and the perceptive centers project it into space as though the object were at some arbitrary distance (*e. g.*, 25 cm.). By the laws of magnification by a simple lens the image is embraced between the lines passing from the optical center of the magnifying lens (the refracting system of the observed eye), through the extremities of the object, and has the size *A'*–*B'*, *A''*–*B''*, etc., according to the distance of projection. In hyperopia rays from *A* and *B* would be divergent, and the observer would have to render these rays parallel by a convex glass or by using his accommodation. In myopia these rays would be convergent, and a concave glass would be required to neutralize their convergence and render them parallel (B. A. Randall).

the same direction after passing through it. This ray is called the *axial ray;* it passes to the corresponding point in the image. Other rays from the same point in the object diverge from the axial ray at various angles; a bundle of these rays is called a *pencil.* The size of a pencil is determined by the diameter of a lens or the aperture of the pupil. The lens gives these unequally diverging rays a direction to a common point or focus. From each point in the illuminated part of the retina a pencil of rays falls upon the crystalline lens and cornea. The size of this pencil equals the diameter of the pupil; to form an image each pencil of rays must be concentrated into one point. By diverging and converging rays is meant the relation the rays from each point bear to each other, not the relation of rays from different points.

Size of the Image.—The details of the eye-ground are considerably magnified in the direct method of examination. In the emmetropic eye the enlargement is found by the following formula: The distance of the retina from the nodal point (optical center) of the eye is 15 mm. The observer projects the image which he sees to the point at which small objects are usually held, say 250 mm. The enlargement of the disk is proportional to these two distances, 15 : 250 : : 1.5 mm. : 25 mm. 16.6 = the enlargement. It is comparable to looking at the disk through a lens of 15 mm. focus, 66 diopters.

It is to be remembered that the farther this image is projected, the larger it appears. In hyperopia the enlargement is less than this. In myopia, on the contrary, it is greater.

Indirect Method.—In the indirect method of ophthalmoscopy a real, inverted image of the interior of the eye is obtained by means of a strong convex lens (object-lens), the principle involved being similar to that of a compound microscope.

The observer holds the object-lens (a convex lens of about 20 diopters) close to the patient's eye, and, placing a convex lens of 5 diopters (eye-piece) behind the ophthalmoscope, throws the light into the pupil and moves his eye nearer to or farther from the patient's eye until he distinctly sees a vessel or a portion of the nerve—that is, a real image of the eye-ground is formed by the object-lens at its focal distance in front of the eye. The observer sees this image, in which all the relations of objects are reversed. His eye is at a distance from the image equal to the focus of the lens in the ophthalmoscope—viz., 20 cm.

The image being inverted, the lower portion of it corresponds to the upper part of the eye-ground, and the right side of the image corresponds to the left side of the eye. If the observer moves upward, the image moves downward; if the observer moves to the right, the image moves to the left. Consequently, the upper part of the image must be viewed if it is desired to see the lower part of the eye-ground, and the right side of the image if parts of the fundus to the left are to be examined.

A comparison between the images as seen by the direct and indirect method may be stated thus: If, in the *direct method* with the disk in view, the observer moves his head to the right, he brings into view a portion of the retina to the left of the disk. The disk now moves out of the field toward the right, and disappears behind the right edge of the pupil. The image, therefore, moves with the observer. If, in the *indirect method* with the image of the disk in view, the observer also moves his head to the right, he sees the image of the same portion of the retina as in the direct method; but this being to the left of the disk, its image occupies a point to the right of that of the disk. The disk thus appears to have moved toward the left. The image, therefore, moves contrary to the movement of the observer's head. Movements in other directions are explained in the same way.

The *formation* of the *inverted image* in ophthalmoscopy may be understood by examining Fig. 51.

In hyperopia and emmetropia a convex lens is necessary to render the rays convergent. In myopia the rays emerge convergent, and the convex lens may be dispensed with in the higher grades, though it is still an advantage because it increases the area of the fundus visible at one time.

Size of the Image.—The enlargement of the image in this method is less than it is in the direct method, but a larger portion of the eye-ground is visible at one time.

The size of the real image of the eye-ground of an emmetropic eye formed by the convex object-lens held at its own focal length from the eye is determined by the following formula: The size of the disk is to the size of the image as the distance from the retina to the nodal point (15 mm.) is to the focal length of the object-glass. If the lens has a focal length of 75 mm., the ratio is 15 : 75; the enlargement is then 5 diameters. A lens of 60 mm. focus would equal an enlargement of 4 diameters—15 : 60.

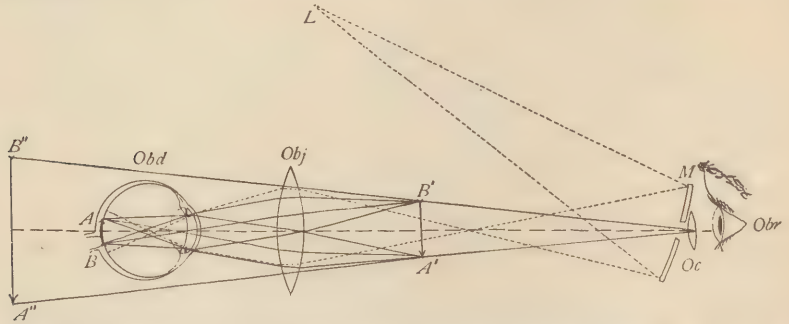


Fig. 51.—Diagram of the indirect method giving an inverted image: Rays from the source of light, *L*, converged toward the observed eye, *Obd*, by the concave mirror, *M*, are intercepted by the lens, *Obj*, and after coming to a focus diverge again and light up the fundus. From a part of the illuminated fundus *A*–*B* rays pass out of the pupil to be again intercepted by the lens *O* and form an inverted real image at its anterior focus *A'*–*B'*. This real image is viewed by the observer's eye behind the sight-hole of the mirror with the aid of a magnifying lens, *Oc*, and is seen enlarged, as at *A''*–*B''* (B. A. Randall).

The observer will see this image under a higher angle in proportion as he comes closer; it will then appear larger. To do this, he must either use his accommodation or place a convex lens (eye-piece) behind the ophthalmoscope. When the eye-piece is used, a virtual image of the aërial image, still more enlarged, is produced, just as in the compound microscope. If the object-lens is withdrawn farther than its focal length from the observed eye, the image in myopia becomes larger, in hyperopia smaller, and in emmetropia remains the same. If the lens is brought closer to the eye, the image becomes smaller in myopia and larger in hyperopia.

Ophthalmoscopy.—The investigation of the deeper structures and interior of the eye by means of the ophthalmoscope may, therefore, be practised with (1) the direct, and (2) the indirect method.

1. **The Direct Method (Method of the Erect or Upright Image).**—The patient should be seated in a darkened room with his back to the

source of illumination—an Argand burner or properly constructed and shaded electric light being suitable—which is placed behind and to the side of his head, on a level with the ear, the face being in shadow, while the rays of light just fall upon the outer canthus of the eye. This will enable the observer to come quite close to the eye without interfering with the path of the illuminating beam. The surgeon sits at that side of the patient which corresponds to the eye under examination—for example, the right—his position being preferably on a slightly higher level than that of the subject. He now takes the ophthalmoscope in his right hand, looks through the sight-hole with his right eye, at a distance of about 50 cm. from the observed eye (the convex border of the instrument being in contact with the concave margin of his brow), meanwhile keeping the other eye open, and reflects the light into the right eye of the patient. If the left eye is to be examined, the ophthalmoscope is held in the left hand.

If an *electric ophthalmoscope* is used, the instrument carries the source of illumination. Moreover, it is not necessary that the room shall be darkened.

If the patient looks directly into the light, the pupil, provided this is not dilated with a mydriatic, will contract, and no satisfactory view is possible. He must hence be directed to turn the head slightly to the right, and gaze into vacancy in the farthest limit of the room, when the pupil will be seen illuminated by a red glare—the reflection from the choroid coat—bright if the pupil is large, and dull if it is small. No details of the fundus are as yet visible at this distance (50 cm.) unless a certain grade of myopia is present or a considerable degree of hyperopia (see page 106).

The beginner should now practise keeping the light steadily in position, and may estimate the success of his endeavor by observing the glare in the pupil. If this changes in color or disappears, the light has shifted from its proper position because the examiner has failed to retain his elbow in close contact with his side, and allowed it to move outward and away from his body, the head meanwhile being bent to one or the other side of the vertical position it should assume in a direct line with that of the subject—feature to feature. This may be understood by observing the two accompanying illustrations (Figs. 52 and 53).

Having gained control of the light, the observer gradually approaches the eye of the patient, taking care that the red glare, which is tinted slightly yellow on the nasal side, marking the position of the optic papilla, remains unaltered, and comes as close as possible—within 1 inch or even nearer. If the maneuver has been successful, and the light directed slightly toward the nasal side, the most prominent feature in the eye-ground—the optic nerve—will come into view; or a retinal vessel may first be manifest, and should be followed to the papilla as a stream would be to its source.

Before proceeding to study the details of the fundus the student should make certain preliminary examinations.

Fig. 52.—Ophthalmoscopic examination. Method of the upright image. Observer and patient in the correct position.

Fig. 53.—Ophthalmoscopic examination. Method of the upright image. Observer in an incorrect position.

(*a*) **Examination of the cornea and lens by transmitted light** is made by placing a + 7 D or 16 D lens behind the mirror, coming close to the eye—that is, until the object to be examined is within the focal

distance of the lens employed, and reflecting the light into the eye in the manner already described.

A foreign body on the cornea, a macula, a deposit on the posterior layer of the cornea, or an opacity in the lens appears as a black object against the red background, in contradistinction to its appearance in its true color under oblique illumination (see page 51).

At the same time the mobility of the iris should be tested, and an observation made as to whether the iris reacts promptly and evenly under the influence of the light directed into the pupil at various angles.

(*b*) **Examination of the vitreous** is made by reflecting the light with the concave or, better, the plane ophthalmoscopic mirror, from a distance of 30 cm. into the eye, while this is moved in various directions so as to bring into view opacities which have a lateral situation or which have sunk to the bottom of the vitreous chamber.

Vitreous opacities and detached retina are seen in the erect position if the observer is sufficiently far away, because they are within his range of accommodation. Small vitreous opacities appear dark; larger ones have a grayish appearance. If he approaches closely, he must place behind the mirror a convex lens, in the manner just described, to bring them into focus, and should always use this method.

(*c*) **Location of Opacities in the Transparent Media.**—If, the observer using the ophthalmoscopic mirror in the manner described in the previous paragraph, an opacity is seen to be freely movable, it must be in the vitreous. Should the opacity move only with the movement of the eye, but not spontaneously, it probably is situated in the cornea or in the lens, although it may be present in the vitreous in the form of a fixed opacity. In these circumstances a differential diagnosis can frequently be made by means of oblique illumination. Should this method prove insufficient, the situation of the opacity may be ascertained by means of its parallactic movement in relation to the border of the pupil. Fuchs states the rule as follows:

The observer looks directly forward into the eye and notes the position of an opacity within the pupillary space. Next, while the patient keeps his eye entirely quiet, the examiner slowly moves from side to side and observes if the opacity retains or does not retain the same position in the pupillary space. If the opacity retains its position unchanged, it lies in the pupillary plane upon or immediately under the anterior capsule of the lens. If it does not retain its original position, it is situated in front of or behind this plane—in front of the plane if the opacity moves in a direction opposite to the direction of the movement of the observing eye, and behind the plane if the opacity moves in the same direction as the observing eye. The quicker the change of position takes place, the farther is the opacity removed from the pupillary plane.

Instead of proceeding in this manner, the observer may retain his position unaltered and cause the patient to move his eye in various directions.

When an opacity is far back in close relation with the retina, its

location may be judged by noting its relation to the movement of the retinal vessels. How far forward it lies in the vitreous may be accurately measured by means of convex lenses (see page 107).

Having ascertained that the media are clear, and having approached sufficiently close, the details of the fundus oculi are brought into view and studied *seriatim*.

If either surgeon or patient is myopic, the necessary concave lens which corrects the error must first be placed in position; while, if hyperopia exists, the fundus is visible without the aid of a glass, provided the hyperopia is not in excess of the power of accommodation.

Beginners, however, often fail to obtain an image of sharp definition, owing to inability to relax accommodation, and succeed in seeing the details clearly only through a concave glass. The power of relaxing the accommodation comes with practice.

The **optic nerve** appears as a nearly round or slightly oval disk, situated toward the nasal side, varying in color from a grayish pink to a more decided red, the tint being most marked upon the nasal half, while the center is occupied by a whiter patch—the "light spot"—marking the position of the entrance and emergence of the retinal vessels. The general tint of the optic disk varies with the age and complexion of the patient and with the intensity of the color of the surrounding eye-ground.

The papilla is bounded by two rings. The outer one, dark colored, usually incomplete or existing only as a slight crescent of pigment upon one or the other side, is the *choroidal ring*, and represents the border of the choroidal coat, where this is pierced by the optic nerve. Within this is a faint white stripe, more distinct in elderly people, the *scleral ring*, which indicates the rim of the sclerotic coat, or, according to Loring, the connective-tissue elements of the inner sheath of the nerve (*connective-tissue ring*). The choroidal ring is often imperfectly produced or entirely absent.

The central white patch may be noticeable only by contrasting it with the color of its surroundings, or it may be a distinct excavation, occupying the center of the disk, and having sharp borders, one of which often shelves slightly outward. This is the *physiologic cup*, and is the space left by the radiation of the nerve-fibers toward the retina, having a floor of white color, because it is composed of the interlacing opaque fibrous tissue, or *lamina cribrosa*, which underlies the optic papilla. It is often stippled in appearance, owing to the lack of light reflected by the non-medullated nerve-fibers, which pass through the spaces of the lamina. According to Schoen, the so-called physiologic excavations are due to dragging of the vaginal processes of the optic nerve and lamina cribrosa from overexertion of the accommodation, and hence are found in adult eyes more commonly than in the eyes of children. They are usually but not always bilateral, and one may be larger than the other. Schweigger traced hereditary transmission in some large physiologic excavations, but doubted if they were associated with any particular refractive condition of the eye.

PLATE I.

The normal fundus of the right eye examined by the direct method of ophthalmoscopy.

The Blood-vessels.—From the central light-spot the *principal retinal arteries* emerge, and into it the *chief venous trunks* empty. Usually one venous and one arterial stem pass directly upward and downward, and on the edge of the disk, or a short distance from it, each divides into two branches. Sometimes this division has taken place in the axis of the nerve behind the lamina, and two arteries and two veins appear directly in the central opening of the papilla, or *porus opticus*. The arteries traverse the surface of the eye-ground, dividing dichotomously into numerous branches, and, passing above and below, spread in greater size and number over the temporal half of the retina, sending small branches toward the macula; and in smaller size and less number over the nasal side. Fine branches arising from the central large trunks, or springing directly from the nerve, pass outward and inward, and also undergo numerous divisions.

The veins pass over the eye-ground in the same general direction as the arteries, and in close relation to them, emptying usually by means of two large branches into the center of the disk.

According to the situation of the vessels, they are named, respectively, upper and lower temporal artery and vein, upper and lower nasal artery and vein, and macular and nasal arteries and veins.

The veins are dark red in color, contrasting with the bright, natural, blood-red color of the arteries. They are slightly tortuous, and larger than the arteries in the proportion of 3 to 2. The difference in color between veins and arteries is most marked in the major branches. In the finer twigs, after four or five divisions, the distinction between arteries and veins is often possible only by tracing them to their source.

Each vessel usually presents a double contour, owing to a bright stripe which passes along the center, leaving a red line on either side. This so-called *light-reflex* has been ascribed to a condensation by the refractive action of the blood column of the rays of light which have passed through the vessel from in front, and have been reflected back slightly from the posterior wall, but chiefly from the underlying tissues.[1] It is more marked upon the arteries than upon the veins, and, indeed, is often absent as the latter cross the disk, being visible in a minor degree when they lie at some distance in the retina.

Pulsation.—The retinal arterial pulse has been classified by Ballantyne as follows: (1) *Locomotor pulse*, that is, a rhythmic displacement of the artery almost synchronous with the cardiac systole; (2) the *expansile pulse*, being a broadening of the blood column; (3) the *capillary pulse*, being a variation in the tint of the optic disk, and (4) the *pressure pulse*, a rhythmic collapse or disappearance of the artery, observed in glaucoma, and produced by pressure on the eyeball, and sometimes called

[1] The cause of the light streak was usually attributed to reflection from the anterior surface of the vessel wall or the anterior surface of the blood column, until Loring maintained that the *refraction of light* was the chief cause of the phenomenon. A. E. Davis endeavored experimentally to confirm Loring's conclusion. Story rejects Loring's theory, and assumes that the reflex comes from the vessel walls. Dimmer ascribes the retinal reflex to the axial blood-stream in the arteries and in the veins to the surface of the blood-stream. Elschnig believes that both in arteries and veins it is the surface of the blood column which causes the reflex.

the *collapsing pulse*. The locomotor pulse is physiologic, at least not pathologic; the expansile pulse may occur in healthy persons, or may be associated with aortic insufficiency; the capillary pulse is seen with aortic regurgitation; the pressure or collapsing pulse is observed in glaucoma, in aortic regurgitation, and in syncope.

Spontaneous pulsation in the veins is a frequent phenomenon. Lang and Barrett found it in 73.8 per cent.; Veasey, in 58.3, and the author, in 62.1 per cent. of their examinations. It may be produced by a slight pressure upon the globe. The spontaneous pulse is due to a communication of the arterial pulsation to the vein, as these vessels lie side by side in the optic nerve, or may be explained by the theory of Donders, that during the systole of the heart (diastole of the retinal arteries) an increased tension in the vitreous is communicated to the walls of the retinal veins, especially the larger ones, at their exit from the eye where the least resistance is offered, obstructing the flow of blood and compressing their lumen. The blood coming from the capillaries overcomes this resistance and the vessels regain their caliber, alternate collapse and distention thus being produced. According to Türk, the venous pulse is due to a continuation of the arterial pulse-wave through the capillaries into the veins.

Physiologic Variations.—The *papilla*, instead of being round or slightly oval, with a vertical long axis, is often distinctly irregular in outline, or has its long axis in a horizontal or oblique direction. Its outer half may be embraced by a crescent of greater or less choroidal changes, the so-called *conus*. A congenital crescent of white appearance, the *underlying conus*, may sometimes be seen below (see page 470), and occasionally is very broad, approximating in appearance a coloboma of the nerve-sheath (see page 469).

The *physiologic cup* varies in size, area, and depth. Normally situated on the temporal side, it may be a deep pit, funnel shaped, with overhanging margins over which the vessels sharply bend, or very shallow and dish-like, sloping to the temporal side, or deep and sharply marked on its inner side, but shading outward.

The *distribution of the vessels* is subject to numerous variations—so much so that it would be difficult to find it the same in any two eyes. The usual departure from the ordinary type is the one already referred to, in which four major branches (two arteries and two veins) appear at the center of the porus, instead of two large branches which later divide at or near the margin of the disk. Anomalies of the veins upon the disk, in the form of unusual bifurcations, are occasionally seen. Division of the vein just before entering the disk; division at the margin; the formation of a vascular circle and final reunion in a single vessel; and anastomosis of the central vein with an aberrant vein, or one which has penetrated the inner side of the disk, have been described (Randall). The veins are normally more tortuous than the arteries. Both sets of vessels present this appearance in marked degree in certain pathologic conditions, but also occasionally as an anomaly without such significance (Fig. 54). Again, the vessels may stand forward from the disk

in a high curve, or twine around each other, as we sometimes see two stems on a vine. Marked enlargement of the anastomoses about the nerve entrance, which anastomoses, according to Leber, connect the retinal and ciliary vascular systems at the level of the papilla and at the level of the choroid, have been described by George Coats.

An anomaly of not infrequent occurrence (7 to 10 per cent. of examined eyes) is a *cilioretinal vessel*, usually, according to Elschnig, an artery, which appears at the temporal border of the disk, then arches outward or away from the papilla, enters the retina, and pursues a general course toward the macula. A large cilioretinal vessel may take the place of one of the temporal arteries. According to Elschnig, a cilio-

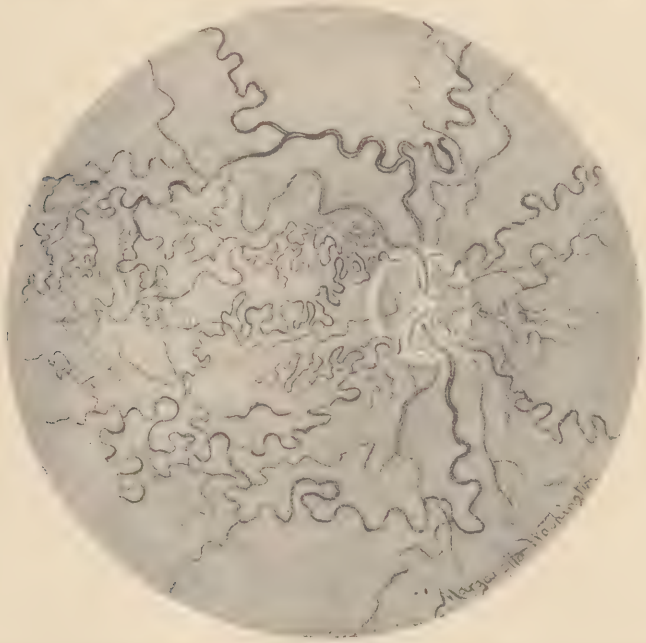


Fig. 54.—Congenital tortuosity of vessels. Note the breadth of the scleral ring.

retinal vessel may be a primary branch of a ciliary artery which pierces the sclera obliquely, without sending a branch to the choroid, and then enters the intrascleral or intrachoroidal part of the optic nerve, or an offset of a ciliary vessel which primarily enters the choroid, where it divides, and one branch passes on into the retina and produces the anomaly in question. Cilioretinal veins may arise from the choroidal vascular system. *Opticociliary vessels* are uncommon. They are practically always veins, only two instances of opticociliary arteries having been reported (Coats). They pass from the central vessels to the disk border, where they disappear under the retina into the choroid. In reporting a case of this character W. T. Shoemaker regards the anomalous vessel as representing an *aberrant choroidal vein*.

The Retina.—Inasmuch as the retina is practically transparent, a study of this membrane is hardly possible without a consideration of the underlying choroid and even the sclera.

In certain persons, especially of dark complexion, the retina assumes a grayish tint in the neighborhood of the papilla, most marked upon its nasal half. This faint opacity is slightly streaked, the striations indirectly corresponding to the expansion of the optic nerve-fibers. Eyes long subjected to the strain of uncorrected ametropia furnish an exaggerated picture of this appearance, which, if at all extensive and associated with similar opacities along the lines of the vessels, assumes pathologic importance (see Retinitis).

In the eye-ground of young subjects, particularly along the line of the vessels, numerous wave-like, glistening reflexes may be seen to follow one after another with the slightest movements of the ophthalmoscopic mirror. The effect is similar to the shimmer seen on the surface of certain silks, and has been designated by English writers *shot-silk retina*. It is unusual to find the phenomenon in individuals over thirty, its occurrence being marked in direct proportion to the youth of the subject.

Macula Lutea.—About two disks' diameter to the outer side of the papilla, and slightly below the horizontal meridian, there is a circular or slightly oval spot, equal in area to the end of the optic nerve, darker in color than the surrounding fundus, uncrossed by any visible retinal vessel, but toward which the finer twigs of the major branches pass, fringing its boundary. This region is the *macula lutea*, or *yellow spot*, and is that portion of the eye-ground concerned with the functions of direct vision.

Its center is occupied by the *foveal reflex*, which marks the edge of the *fovea centralis*, and which may appear as a spot of light, a small circle with reddish center, a shifting crescent, or a shining line. This, in turn, is surrounded by a dark area (the dark spot of the macula), sometimes containing a number of brownish-black or light colored or even glistening granules, often called *Gunn's dots*, which have no pathologic significance. Finally, the margin of the macula is bounded by a glistening *whitish ring* or *halo* (macular reflex).

The method of examination determines whether all these characteristics of the macula lutea can be observed. They are fairly constant, however, with the exception of the halo, and are notable in young children. Ordinarily, the macular ring is best seen with the indirect image in young eyes, where it is apt to assume an oval shape; but according to Lindsay Johnson, even with the upright image, if the source of illumination be gradually lowered, a time is arrived at when more light is reflected from the macula than from the general fundus, and at that moment the ring appears. In elderly people the region usually cannot be well recognized except by the absence of vessels and its darker color, but even in them careful focusing will not infrequently reveal the foveal reflex. In albinos it is still more difficult to define this area.

Although no vessels visible to the ophthalmoscope cross the macula, except as an anomaly (Randall, Johnson), the region is abundantly

supplied with capillaries, which can be shown by artificial injection, which surround the fovea in a close loop, but do not occupy it. The student may find the region difficult to study because the light falling upon it causes the pupil to contract, the view being further hindered by the corneal reflex. Hence the pupil should be dilated, when the macula may be brought into view by requiring the patient to look directly into the ophthalmoscopic mirror, or may be found by turning the light outward from the lower edge of the disk. The region should always be studied with the utmost care.

The appearances in the macula depend partly upon the disposition of the layers of the retina in this region. At its margin the retina is much increased in thickness by an extra development of the layer of the ganglion cells, while the fovea is produced by the hollowing out of the center of the macular region. The macular reflex, or ring, therefore, may be considered as a reflection arising from the thickened macular circumference, and the foveal reflex as a reflection from the edge of the fovea. The variations, according to Johnson, are due to the direction and the shape of the sloping sides of the pit, but, according to Dimmer, depend upon the kind of ophthalmoscopic mirror which is employed, the reflex being the inverted image of the center of the mirror. According to Piersol, the color of the macula depends upon the presence of a yellowish pigment within the layers internal to the visual cells, the latter elements remaining colorless; in consequence of this arrangement the fovea, in which the neuro-epithelium alone exists, is devoid of pigment, and, therefore, appears as a light spot within the colored area. The dark-brown spot of the macula is generally believed to depend upon the thinning of the retina at this spot, with a more decided pigmentation in the epithelium. Dimmer, however, thinks that it is also produced by absence of the slight veiling of the retina at this point, which is manifest in the surrounding more compact layers.

The Choroid.—The bright glare which illuminates the pupil when the light is thrown into it from the ophthalmoscopic mirror, and develops into the uniform red color of the fundus, when this is brought into view, arises from the choroid. The rays of light pass through the transparent retina to its pigment epithelium, which in ophthalmoscopic work is accredited to the choroid, and in part are absorbed and in part reflected. The greater the quantity of the pigment, the greater the amount of absorption, so that the color of the eye-ground depends upon the degree of saturation in this epithelium, and varies from an almost slaty color in the dark-skinned races to a dark red in persons of blond complexion. A light yellowish-red or brownish color is often evident.

In very fair people the imperfect development of pigment-cells of the choroid exposes the larger choroid vessels, which are evident as a meshwork of tortuous red bands with intervening spaces of lighter or darker color, and which are distinguishable from the retinal arteries and veins by their flat appearance and absence of the light streak. A nearly perfect exposure of the choroidal vessels is seen in albinos. It is not usually possible with the ophthlamoscope to differentiate the arteries

and veins of this system, although the latter are of greater size, and, near the equator of the eye, converge toward the venæ vorticosæ, being separated by larger and longer spaces. In decided brunettes these spaces are more deeply tinted than the vessels, which appear "like light streams separated by dark islands" (Nettleship). A fair general idea of what tint may be expected in the fundus may be obtained by observing the color of the patient's hair.

All the details of the eye-ground may be studied with greater ease through a dilated pupil, and, on beginning his studies, the student may with propriety employ a mydriatic—euphthalmin, cocain, or homatropin, not atropin—provided no signs of glaucoma are present Having acquired a knowledge of the normal appearance thus seen, he must now practise with the undilated pupil.

The disk and macula having been studied, the peripheral parts of the eye-ground should be examined by throwing the light inward, upward, and downward, the head of the observer being moved correspondingly to comply with the changed direction of the mirror. Even when the central part of the fundus presents the usual characteristic red tint, the choroidal vessels are frequently exposed in the periphery, presenting the appearance just described, and having no pathologic significance.

Determination of Refraction by the Ophthalmoscope.—The estimation of the refraction of the eye by means of the ophthalmoscope results in either a *qualitative* or a *quantitative* determination.

The former is obtained in the following manner: Hold the ophthalmoscope 30 to 50 cm. from the patient's eye, and, looking through the central aperture of the mirror, unaided by a glass, observe if any vessels come into view. Their appearance means that the eye is either hyperopic or myopic. Now move the head from side to side, and note if the vessels move apparently in the same or in a direction opposite to the movements of the head. If the former, the eye is hyperopic; if the latter, myopic. Inasmuch as the image of the vessels in low degrees of myopia would be formed only at a considerable distance from the observed eye (30 to 120 cm.), and since no sharp image would be obtained in either emmetropia or low degrees of hyperopia farther away than 30 cm., any considerable degree of ametropia may be excluded by failure to obtain a direct image except at a long range or a very short distance from the patient's eye.

Before attempting a *quantitative* estimation of refraction by means of the ophthalmoscope, certain fundamental rules must be observed:

1. Both surgeon and patient must have relaxed accommodation.

2. A certain definite spot in the eye-ground upon which to focus should be selected.

3. The observer should approach as close as possible to the eye under observation.

4. In order to ascertain correctly the refraction error, the observer must be emmetropic, or, if not, render his eye emmetropic by using the proper correcting lens, in the form either of spectacles or of an equivalent glass placed behind the sight-hole of the ophthalmoscope.

The emmetropic observer can see the details of the myopic eye-ground only dimly without the aid of a correcting glass, and not at all if the myopia is of high degree. By placing concave glasses behind the sight-hole of the ophthalmoscope the convergent rays which leave the observed eye are rendered less and less convergent, until that glass is reached which just yields a distinct image—*i. e.*, one which has rendered the convergent rays parallel.

The emmetropic observer can see the details of a hyperopic eye-ground distinctly without the aid of a correcting glass, unless the hyperopia is of very high degree, by an effort of accommodation which renders his crystalline lens more convex, and thus causes the divergent rays which leave a hyperopic eye to become parallel. But, with accommodation relaxed, he sees distinctly the details of the fundus through a convex lens placed behind the ophthalmoscope; this should be substituted for other stronger convex lenses until the strongest one is reached with which a clear image is still possible—*i. e.*, one which has rendered the divergent rays parallel, while the next highest number creates a blur over the details of the eye-ground.

From what has been said it follows that the strongest convex lens, placed in position in the ophthalmoscope, with which the emmetropic observer can still see the details of the fundus at the point selected measures the degree of hyperopia; the weakest concave lens, the degree of myopia. The hyperopia usually is somewhat greater, and the myopia somewhat less, than the result obtained by ophthalmoscopic examination.

In order to estimate the refraction of the eye examined, the hyperopic observer must subtract from the convex, or add to the concave, lens, which yields him a sharp image of the fundus, the amount of his own error, while the myopic observer must add to the convex, or subtract from the concave, lens, with which he sees the eye-ground the degree of his own near-sightedness.

In order to calculate the amount of lengthening or shortening of the eye equal to a lens which neutralizes the myopia or hyperopia in any given case, and provided the distance between the surgeon's eye and that of the patient is not more than 2.5 cm., the following table, which was prepared by the late Mr. Nettleship, is useful:

Hyperopia of	1	D represents a shortening of	.....	0.3	mm.
"	2	" " "	.....	0.5	"
"	3	" " "	.....	1	"
"	5	" " "	.....	1.5	"
"	6	" " "	.....	2	"
"	9	" " "	.....	3	"
"	12	" " "	.....	4	"
"	18	" " "	.....	6	"
Myopia of	1	D represents a lengthening of	.....	0.3	"
"	2	" " "	.....	0.5	"
"	3	" " "	.....	0.9	"
"	5	" " "	.....	1.3	"
"	6	" " "	.....	1.75	"
"	9	" " "	.....	2.6	"
"	12	" " "	.....	3.5	"
"	18	" " "	.....	5	"

By this table the depth of an excavation in the papilla may be measured. For instance, if the bottom of the pit required − 5 D for its sharp examination, and the margin of the nerve was seen without any glass, the depth of the excavation would be 1.3 mm.

The presence of *astigmatism* may be ascertained by means of the ophthalmoscope and the upright image because all points of the portion of the fundus under examination are not in focus at the same time—*e. g.*, the retinal vessels running in the directions which correspond to the principal meridians.

Thus, when two vessels cross each other at right angles, the vertical branch may be sharply seen, while the horizontal one presents a blurred image, or the upper and lower margins of the disk may be clear, but the lateral borders indistinct. The amount of hyperopia or myopia of the *vertical* meridian is equal to the strongest convex, or weakest concave, glass which makes distinct the vessels running in a *horizontal* direction. The refraction of the *horizontal* meridian is determined by the glass which yields a clear image of the vessels running in a *vertical* direction. As the vessels do not correspond to the layer of the rods and cones, the measurement is an approximation.

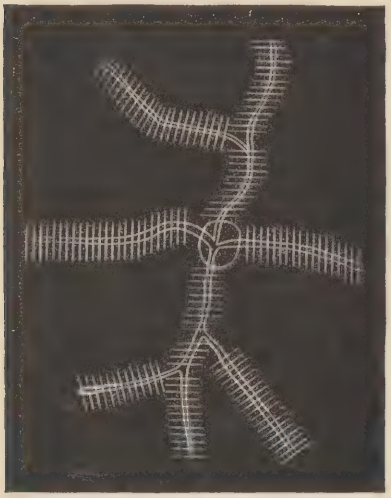


Fig. 55.—Focusing of the vessels by the meridians of an astigmatic eye; the parallel lines on each vessel represent the direction of the meridians through which a distinct image of the vessel is obtained.

Compound astigmatism is determined by finding, in hyperopia, the strongest convex lens which the vessels in each meridian will bear with the preservation of a distinct image, and subtracting the one from the other, thus finding the difference between the meridians—*i. e.*, the amount of astigmatism. In high degrees of astigmatism the optic disk usually appears as an ellipse, its long axis corresponding with the meridian of greatest refraction.

The measurement of astigmatism in this manner, with any degree of accuracy, requires much practice, a perfect control of the accommodation, and even then must never be employed to the exclusion of other and more trustworthy methods.

2. **The Indirect Method** (**Method of the Inverted Image**).—The patient and surgeon are seated in the same relative positions as have already been described in connection with the direct method, and, if the right eye is to be examined, the ophthalmoscope is held in the right hand at a distance of 30 cm. from the patient, who is instructed to look at the right ear of the examiner. A convex lens of 20 D, held between

the surgeon's left thumb and index-finger, while the remaining fingers are rested upon the brow to steady the hand, is placed at about its own focal length in front of the patient's eye, directly in the path of the rays returning from the fundus, which are thus brought to a focus and form an aërial image between the observer and the glass.

If the left eye is to be examined, the ophthalmoscope is held in the left hand, and the patient instructed to look at the surgeon's left ear, while the lens, grasped in the fingers of the right hand in the manner just described, is placed in position.

The image which is found at a certain distance in front of the object-glass may not present itself to the observer as a distinct picture, owing to his inability to accommodate for the point of its formation. This accommodative strain may be relieved and the image magnified by

Fig. 56.—Method of an indirect examination with the ophthalmoscope.

placing behind the ophthalmoscope a convex glass of 5 D, which adapts the emmetropic observer, with relaxed accommodation, for a point 20 cm. distant. If the observer is presbyopic, or has a deficient amplitude of accommodation, this additional lens is absolutely necessary; while if he is hyperopic, the degree of his hyperopia should be added to the glass used as a magnifier. The observer possessing a moderate degree of myopia requires no lens in the ophthalmoscope, because he views the aërial image at his far point, while if his myopia is of high grade, he will need a weak concave glass.

With the indirect method of examination the field is larger than in the direct method. Although individual objects in the field are small and sharply defined, the details of the fundus are less perfectly revealed than with the direct method. In young subjects in the macular region a bright reflex encircles an elliptic dark area containing in its center a

reddish or, less frequently, a bright point surrounded by a small brilliant ring. These characteristics are not always present.

A *qualitative* estimation of the refraction with the indirect method may be ascertained with the mirror alone, in the manner already described (see page 108). Furthermore, ametropia of high degree may be recognized by varying the distance of the object-lens from the eye. Withdrawal of the lens from the eye causes the image to appear smaller in hyperopia, larger in myopia.

The measurement of the degree or quantity of the refraction and the estimation of astigmatism by the indirect method were at one time much practised, but in practical work the methods are so far inferior to those usually employed (skiascopy, ophthalmometry) that their description is omitted.

Ophthalmodiaphanoscopy.—With the ophthalmodiaphanoscope, designed by Carl Hertzell, it is possible to make examinations of all parts of the eyeball and of the orbit around and posterior to the globe. Dr. H. F. Hansell thus describes the instrument: It consists of an 80-candlepower electric lamp, strengthened by a reflector, which increases the power of the light to 100 candlepower. The lamp is held by the patient far back in his mouth and a black mask is adjusted to protect the face. The anterior rays pass through the buccal plate of the superior maxillary bone, through the antrum and its roof to the floor of the orbit; the posterior rays pass through the hard and soft palate, the lateral walls of the nose, the anterior cells of the sphenoid, and pass to the median orbital walls. The eye-ground is illuminated from behind, below, and from the median side. The observer approaches the lighted pupil as near as possible and examines the illuminated fundus without the aid of any other instrument. If the eyes are ametropic (myopic) a correcting glass should be worn, and Hansell finds mydriasis advantageous. According to Hertzell, the optic nerve appears more opaque than it does in ordinary ophthalmoscopic examination, its outlines are sharply defined, and if a pigment ring exists, it is distinct. The retinal veins are very dark, the arteries somewhat lighter in color; the macula appears as a dark spot. Retinal hemorrhages appear as dark, sharply defined areas. A tumor would diminish the illumination and obscure the fundus details. The brilliant transillumination of the sinuses and orbit is instantly obstructed by the presence of a growth, thickening, or opacity, and the diagnosis thereby facilitated. The author's experience with the instrument is limited to a few observations made with Dr. Hansell; it seems to be of distinct value in the diagnosis of orbital and sinus disease.

Ophthalmometry.—This term indicates mensuration of the eye, and, as usually employed, is limited in its application to the measurement of the radius of curvature of the cornea (*keratometry*). In order to practice ophthalmometry, instruments for taking the measurement of the radius of curvature of the cornea have been constructed, and are known as *ophthalmometers*. The ophthalmometer most in use is the one devised by Javal and Schiötz.

Other instruments are those designed by Leroy and Dubois, Reid, Hardy, Chambers-Inskeep, and Sutcliffe. In the opinion of the author, a suitable ophthalmometer, or, more accurately, keratometer, is of the greatest service in determining the refraction of the cornea and the direction of its principal meridians. None of these instruments should be used to the exclusion of other methods, especially the employment of mydriatics and skiascopy. (For a full description of the method of using the ophthalmometer see Appendix, page 687.)

Optometry is a term which indicates the principles involved in the measurement of the refraction of an eye by its limits of distinct vision. The instrument which thus serves to determine the refraction of the eye is called an *optometer.*

Optometers are based upon a number of principles. For instance, a single convex lens by which the direction of the luminous rays emanating from an object is changed, and consequently the determination of the refraction of the eye rendered possible, constitutes an optometer. Other optometers are based upon the principle of a telescope; still others upon the measurement of circles of diffusion, upon Scheiner's experiment, and upon the chromatic aberration of the eye. It would not be possible, in the limits of this manual, to describe in detail the principles involved or the various forms of apparatus which have been employed. Should the student desire to pursue the subject, he may with advantage consult the chapter devoted to this method found in Landolt's *Refraction and Accommodation of the Eye.*

Of the many instruments constructed in recent times for the purpose of estimating the refraction of the eye, and to which the name *refractometer* is usually applied, the best is the one devised by the late Dr. William Thomson.[1]

Skiascopy, or the Shadow-test (Retinoscopy).[2]—This is a method of determining the refraction of the eye by observing the direction in which the light appears to move across the pupil, when it is made to move back and forth across the face by rotation of the mirror which reflects it to the eye.

With the ophthalmoscope, as has already been explained, the observer may look into a myopic eye from close in front of it and see an erect image of the fundus, which he can render clear by the proper concave lens; or, in the same eye, from a greater distance, he can view an inverted image of the fundus, with or without the intervention of a convex lens. The point at which the change from the erect to the inverted image occurs has been called the *point of reversal.* It is the point for which the eye is focused, and is the far point of distinct vision. Skiascopy is simply an accurate method of determining this point of reversal.

To apply the test with the plane mirror the surgeon faces the patient at a distance of about 1 meter or less; and, holding the mirror to his own eye, reflects on the patient's face the light from a lamp placed near

[1] Transactions of the American Ophthalmological Society, 1902, vol. ix.
[2] This section has been prepared and revised by Dr. Edward Jackson.

the mirror, and covered with an opaque shade having an aperture 3 to 6 mm. in diameter. By rotating the mirror the area of light it throws on the face is made to move up and down, or from side to side, or obliquely. The part of the light that falls on the patient's pupil is condensed on his retina, forming there a small light area which also moves as the mirror is rotated; for the plane mirror this retinal light area always moves in the same direction as, or "with," the light on the face.

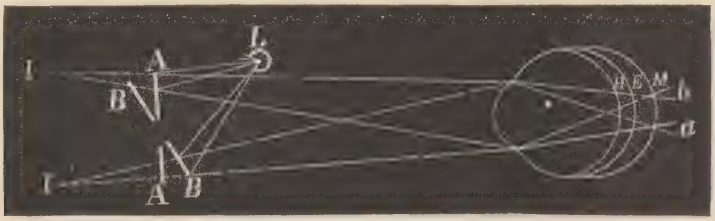


Fig. 57.—Skiascopy with the plane mirror.

In Fig. 57 L represents the lamp-flame, screened from the patient, and A and B two positions of the plane mirror. When the mirror is at A, the light that enters the eye will come as though from a flame at l, and will be condensed toward a, on the lower part of the retina. At this time the light falls on the lower part of the face. But when the mirror is rotated to B, the light entering the eye comes from the direction l', and is condensed toward b, on the upper part of the retina. At the same time the light on the face moves upward. The positions of the retina in hyperopia, emmetropia, and myopia are shown at H, E, and M.

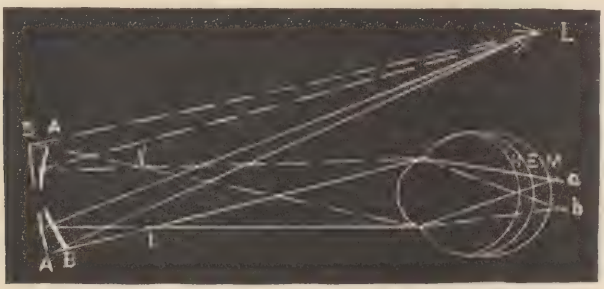


Fig. 58.—Skiascopy with the concave mirror.

It will be noted that in all these forms of ametropia the movement of the light on the retina is *with* the light on the face. When skiascopy is practised with a concave mirror, the lamp-flame which serves as a source of light must be placed behind the patient; and the light area on the retina moves in an opposite direction "against" the light on the face, "against" the movement of the mirror.

In Fig. 58 the action of the concave mirror is represented. When the mirror is at A, the light that enters the eye comes from the focus of the mirror at l, conjugate to the position of the lamp-flame, and is

condensed toward *a*, on the upper part of the retina; and when the mirror is at *B* the light enters from *l'*, the new position of this conjugate focus, to be condensed toward *b*, on the lower part of the retina—that is, as the light has moved upward on the face, it has moved downward on the retina, and this is true for either *H*, *E*, or *M*.

The following account assumes the use of the plane mirror, but will apply equally for the concave mirror, if one bears in mind that with the latter the movement in the pupil is always in the opposite direction, and that the lens before the patient's eye must be changed, instead of changing the surgeon's distance from the patient (see page 116).

We have thus seen what is the *real* movement of the light on the retina, as it would appear in the back of an enucleated eye with the sclera and choroid removed, but the surgeon does not see it in that way; he can only watch the *apparent* movement as seen through the pupil. This will be the same as the real movement, with the light on the face [plane mirror] when he sees an erect image, and in the opposite direction when he sees an inverted image.

In Fig. 59 *M* represents a myopic eyeball, from the retina of which rays come out and are focused at *B*, the *point of reversal*. Anywhere

Fig. 59.—Rays coming from a myopic eyeball.

closer to the eye than this, as at *A*, an erect image is seen; the light in the pupil seems to move *with* the light on the face. Anywhere beyond the point of reversal, as at *C*, an inverted image will be seen, and the light in the pupil will appear to move *against* the light on the face (see page 165). Just at the point of reversal *B* it is impossible to see which way the light moves, and the illumination of the pupil is very feeble.

At one or two diopters from the point of reversal the light is comparatively bright. As the examiner goes farther than this from the point of reversal, it becomes more and more feeble. With the same movement of the mirror the apparent movement of the light in the pupil is quicker as the point of reversal is approached. These variations in the degree of illumination and rapidity of movement may aid the expert in choosing the lens to be next placed before the eye, but the thing mainly depended on is the direction of the movement.

Application in Myopia.—If the surgeon, on throwing the light into the eye, finds that its apparent movement in the pupil is *against* the light on the face, he must be farther from the eye than the point of reversal (*B*, Fig. 59). He should then slowly approach the patient, still rotating the mirror and watching the apparent movement of the

light, until he finds this apparent movement is *with* the light on the face, as at *A*. He is now closer to the patient than the point of reversal, and should draw back and observe the greatest distance (*A*) at which this movement with the light on the face can be distinguished; then, drawing farther back, he observes the nearest point to the eye (*C*) at which the inverted movement can be seen, and the point *B*, half-way between *A* and *C*, is to be taken as the point of reversal. These observations should be repeated until the exact position of *B* is established. The distance from *B* to the eye is then measured; it is the focal distance of the glass required to correct the myopia. For instance, if the erect movement is seen as far as 55 cm. from the eye, and the reversed movement as near as 80 cm., the point of reversal will be about 67 cm., and the myopia, therefore, 1.50 D.

If the myopia thus discovered is high, its amount can be most accurately determined by putting on a concave lens that will correct all of it but 1 or 2 D, measuring what is left by skiascopy, and adding this to the strength of the lens used to get the total myopia.

If, on the other hand, the myopia is very low, the point of reversal may be at so great a distance that when near it one cannot see which

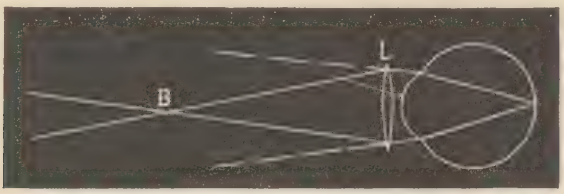


Fig. 60.—Rays emerging from a hyperopic eye.

way the light is moving in the pupil. In this case a weak convex lens must be placed before the eye, the point of reversal found with the lens, and then the strength of the lens deducted from the myopia which this indicates in order to find the myopia of the eye.

Application in Hyperopia.—Here the rays from the retina emerge divergent, as shown by the broken lines in Fig. 60, and there can be no point of reversal anywhere in front of the eye. The surgeon finds the apparent movement of the light in the pupil is *with* the light on the face, and it continues to be so, no matter how far he draws back. It is necessary, then, to place a convex lens (*L*) before the eye strong enough to render the rays convergent, and so to make a point of reversal a convenient distance in front of the eye. This lens does two things: First, it overcomes the divergence of the rays; this takes part of its power. Second, the remainder of its power makes the rays converge, causing a sort of artificial myopia. The point of reversal (*B*) obtained is the point of reversal for this artificial myopia. It is to be determined as for natural myopia, and the amount of myopia it represents deducted from the total strength of the lens. The remainder will be the power required to overcome the divergence of the rays, or the strength of lens needed to correct the hyperopia.

For example, suppose the movement of the light in the pupil is found at all distances to be *with* the movement of the light on the face, and on placing a 5 D convex lens before the eye it is found to be still *with* the movement of the light on the face when the examiner approaches to a little within 1 meter, but appears reversed if looked at from a distance slightly greater than 1 meter. The point of reversal then is at 1 meter; 1 D of the strength of the lens is making the rays convergent, while the other 4 D have been used to overcome the divergence of the rays as they came from the eye. Therefore the eye must be 4 D hyperopic. For accuracy it is better here, as in the case of natural myopia, to make the final determination with a lens that brings the point of reversal $\frac{1}{2}$ to 1 meter from the eye.

Application in Emmetropia.—The application of skiascopy for emmetropia is precisely the same as for hyperopia; but it is found that the artificial myopia caused by the convex lens equals the full strength of the lens, proving that the rays must have emerged from the eye parallel.

Application in Regular Astigmatism.—The principles involved and the methods to be employed are essentially the same as in myopia or hyperopia; but the refraction has to be determined in the two principal meridians instead of in any meridian indifferently, as it can be where all meridians are alike. To determine the refraction in a certain meridian the light must be made to move back and forth in that particular meridian by rotating the mirror about an axis at right angles to it.

The direction of either of these principal meridians is revealed by the area of light in the pupil assuming the form of a more or less distinct *band of light*, extending across the pupil in the direction of this meridian, when its point of reversal is approached. This band can be clearly distinguished only when the surgeon's eye is much nearer to the point of reversal for one principal meridian, than to the point of reversal for the other principal meridian. In such a position this band is, for the higher degrees of astigmatism, very noticeable, and fixes with the greatest accuracy the direction of the principal meridian. When the band-like appearance is most noticeable, it is easy to cause its apparent movement from side to side; but it is more difficult to distinguish the movement in the direction of the length of the band. Still, this latter movement is the one that must be especially watched, and its reversal point determined.

When the astigmatism is very low, the appearance of a band may be very indistinct, or not at all perceptible. But in such cases it will be found that when the surgeon has reached the point of reversal for movement of the light in one direction, there is still distinct movement, either direct or inverted, in the direction at right angles to this; and he will thus know he has tested one meridian of an astigmatism, and must in the same way ascertain the point of reversal for the other at right angles to it. When the surgeon is closer to the eye than the point of reversal for either meridian, the movement will be *with* the light on the face *in all*

directions. When he is at the point of reversal for the meridian which has its point the nearer to the eye, there will be *no distinguishable movement in the direction of the band* here visible, but still a *movement* (*with*) *at right angles to it.* When he is between the two points of reversal, there will, in the direction of the nearer meridian, be an inverted movement of the light (*movement against*), but in the other meridian a direct movement (*movement with*). When the farther point of reversal is reached, the *direct movement* in its meridian *ceases*, while the movement in the other meridian continues inverted (*against*). When the surgeon has drawn back beyond both points of reversal, the movement is reversed, *against the light on the face in all directions.*

Having determined the amount of myopia, natural or artificial, in both principal meridians, the strength of the cylinder required to correct the astigmatism will, of course, be the difference between the refraction for the two meridians. Having thus ascertained it, it is well to put this cylinder before the eye and to see if it does accurately correct the astigmatism, giving the same point of reversal for all meridians of the cornea; and, for accuracy, the spheric lens which will bring this point of reversal to the distance of $\frac{1}{2}$ to 1 meter should be used with it.

Application in Irregular Astigmatism.—If the pupil is dilated, it will always be found that the refraction of the eye varies in different parts of it, so that points of reversal for different parts of the pupil lie at different distances in front of the eye; and at the point of reversal and near it, both direct and reversed movements of the light are visible at the same time in these different parts of the pupil. Usually there is at the center of the pupil a considerable area that has about the same point of reversal, called the *visual zone.* This is the part through which light will come to be focused on the retina when the eye is in use. For practical purposes it is to the refraction of the visual zone that attention should be paid, the refraction in the other parts of the pupil being of little practical importance. On account of the small size of the visual zone in many eyes it is best to apply skiascopy from a distance of less than 1 meter from the patient's eye.

When the visual zone of the pupil differs materially in refraction from the part of the pupil that surrounds it, the eye is said to present *aberration.* This is called *positive* when the center of the pupil is more hyperopic or less myopic, and *negative* when the opposite is the case. When the aberration is high, on examining it from near the point of reversal of the margin of the pupil, the movement of the light will be swift at the margin and slow in the center, making it look as if the light in the pupil were wheeling around a fixed point at the center. This appearance is marked in conical cornea. Aberration of moderate degree causes the appearance of a ring of light at the margin of the pupil, which has a very distinct movement when the point of reversal for the center of the pupil has been reached.

The Concave Mirror.—With the concave mirror the movement in the pupil is reversed (see page 113); and one cannot vary much the distance of the mirror from the patient's eye, but must keep a fixed

distance (usually somewhat less than 1 meter), and bring the reversal to this point by changing the lenses used before the eye.

The Use of Mydriatics.[1]—In addition to the use of the mydriatics in the treatment of diseases of the eye—*e. g.*, iritis—these drugs are employed as aids of an accurate determination of ametropia. With the ophthalmometer, without the aid of mydriatics, and with the method of skiascopy, in the absence of prolonged mydriasis, good results are obtained; but in all patients of suitable age, and in the absence of contra-indicating symptoms, an active mydriatic should be employed in the measurement of errors of refraction, especially to detect astigmatism. The mydriatic accomplishes three purposes:

1. It dilates the pupil, and permits a thorough exploration of the interior of the eye, as well as a more perfect examination of the lens and vitreous humor than could be obtained without its aid. The student should not, of course, think it necessary to dilate the pupil of each eye which he subjects to an ophthalmoscopic examination; but glasses should not be adjusted without a thorough knowledge on the part of the examiner of all the details of the eye-ground and the transparent media.

2. It paralyzes the action of the ciliary muscle and places the accommodation in abeyance, rendering manifest types of ametropia which otherwise would remain latent.

3. It fulfils the important function of giving, during the time of its action, physiologic rest to the eye that is under its influence, and consequently helps to subdue any retinochoroidal disturbance or other congestive condition that pre-existing eye-strain may have originated.

In practice, various mydriatic (cycloplegic) drugs are employed, the most common being the sulphates of atropin, hyoscyamin, and duboisin, and the hydrobromate of homatropin and scopolamin.

(*a*) *Atropin.*—Atropin sulphate is usually employed in a strength of 4 grains (0.26 gm.) to the ounce (30 c.c.). A drop of such a solution dilates the pupil in about fifteen minutes, and a very few moments later begins to paralyze the accommodation, which sustains a full paralysis in about two hours. The effect of atropin upon the accommodation remains for a week, but if, as is commonly the case, the drug is used for several days at a time, this influence is much prolonged, and full return to the previous power of accommodation is not secured for about twelve or fourteen days.

In using atropin sulphate for the purpose of correcting errors of refraction, a solution of the strength given above should be instilled into the eye, one drop at a time, three times for at least a day, preparatory to the determination, and in young subjects possessing hyperopic eyes, with active ciliary muscles, especially if there is associated spasm of accommodation, the drug must be continued for several days, or even longer, before the desired result is reached.

(*b*) *Hyoscyamin* is usually employed in the strength of 2 grains (0.13 gm.) to the ounce (30 c.c.), in the same manner. It produces

[1] In place of the term "mydriatic" the word "cycloplegic" is sometimes employed if, in addition to the mydriasis, there is paralysis of accommodation.

wide dilatation of the pupil and complete ciliary paralysis, the effect of which lasts from six to seven days. Many surgeons prefer this drug to atropin, and believe that its effects are equally good, while it enjoys the advantage of a much more temporary action upon the function of the ciliary muscle. The salt must be neutral, and the solution filtered through neutral paper (Risley).

(*c*) *Hyoscin* and *duboisin* in similar strength have similar actions, the latter drug being even more transitory than hyoscyamin in its effect, return to accommodative power occurring in from four to five days. Both of them have the disadvantage of producing marked constitutional disturbances, at times rendering their employment disadvantageous. Hyoscin is chemically and physiologically identical with scopolamin.

(*d*) *Homatropin* is a drug which produces a transitory effect upon the ciliary muscle, full return of accommodation occurring in about fifty hours after the last instillation.

To use this drug properly it must be employed by cumulative instillations in the strength of 8 to 16 grains (0.52–1.04 gm.) to the ounce (30 c.c.), one drop of such solution being used every fifteen minutes for an hour and a half preceding the determination, and then waiting forty minutes. At the end of this time the maximum effect of the drug upon the accommodation is secured. In the opinion of some surgeons this drug is an insufficient paralyzer of accommodation, but if caution in regard to the cumulative instillations is observed, and the rule given above carefully followed, entirely satisfactory results may be obtained. Its influence may be neutralized by eserin. Some surgeons prefer homatropin in gelatin disk form, associated with cocain, in the determination of errors of refraction. The author has never been able to convince himself of their superiority to a solution of the drug, and regards the addition of cocain to the solution as a distinct disadvantage.

(*e*) *Scopolamin*, introduced by Raehlmann, may be employed in the strength of 2 grains (0.13 gm.) to the ounce (30 c.c.). Two instillations of one drop each forty-five minutes apart are sufficient. Mydriasis begins in twelve, and is complete in thirty, minutes; cycloplegia occurs in about forty-five minutes. Full return of accommodation may be expected in from five to six days. Toxic symptoms—staggering, vertigo, and dryness of the throat—may develop. Scopolamin is said to be more valuable than atropin in inflammatory affections of the eye, and not to increase intra-ocular tension (Raehlmann).

It is not safe to use strong mydriatics in elderly persons, and they must never be employed if there is any symptom of glaucoma. They are usually unnecessary when that age has been reached after which the accommodation is so weakened that hyperopia ceases to be latent, and they are rarely employed after the forty-fifth year; but Hess and Duane have demonstrated that hyperopia may be as latent at the age of forty-five or fifty as it is in young persons, and, therefore, the need of a mydriatic may be equally important.

Euphthalmin is an active mydriatic in a 5 to 10 per cent. solution.

It produces maximum dilatation of the pupil in about fifteen or twenty minutes, and the pupil returns to its normal size in five to six hours. Its influence on accommodation is so slight that it has no practical value as a cycloplegic. It is an admirable agent for producing brief dilatation of the pupil, and, fortunately, it has no perceptible effect upon the cornea. It may be combined with cocain, 1 per cent. of each, and its mydriatic efficiency thereby enhanced.

Hydrochlorid of cocain, in addition to its anesthetic action, is, in 2 to 4 per cent. solution, an excellent mydriatic, but its effect upon the accommodation is so slight that it is valueless for the purpose of preparing an eye for the estimation of any error of refraction.

Other mydriatic drugs which may be mentioned are *ephedrin homatropin*, 1 : 10 (Groenouw); *mydrol*, 10 per cent.; *methylatropin*, 5 per cent., and *atroscin*. The last-named drug is similar to scopolamin in its action.

CHAPTER IV

NORMAL AND ABNORMAL REFRACTION

The cornea, aqueous humor, crystalline lens, and vitreous body are the media by which rays of light passing into the eye are refracted and brought to a focus with the production of an image on the retina. Because the two surfaces of the cornea are practically parallel and the index of refraction of the cornea and the aqueous humor are the same, the *dioptric apparatus* may be reduced to the anterior surface of the cornea and the anterior and posterior surfaces of the crystalline lens. The cornea is the principal lens when the eye is at rest, and it has a higher refractive power than the crystalline lens; but during maximum accommodative effort the refractive power of the crystalline lens approaches that of the cornea. The formation of a distinct retinal image requires that the curvature of the corneal meridians shall be symmetric, that the plane of the lens shall be perpendicular to the visual line, that its sectors shall have a uniform density in corresponding layers, and that the focal length of the dioptric apparatus shall correspond with the length of the visual axis.

Emmetropia.—To the normal eye, which produces a distinct image of distant objects on the retina, without accommodative effort, the term *emmetropic* is applied, and the condition may be defined as follows:

Emmetropia is that refractive condition of the eye in which the visual axis corresponds exactly with the focal length of the dioptric apparatus when at rest; the far point lies at infinity, and the eye, in its condition of minimum refraction, is adapted to focus parallel rays on the retina. The principal focus lies on the retina.

The emmetropic eye has an average length of about 22 mm., although emmetropia is still possible with a longer or shorter axis, if the curvature of the ocular lenses varies in proportion. Emmetropia, although it exists but rarely, is the ideal state of refraction. Such an eye has a range of vision from infinity to its near point (see table, page 39). Glasses are not required for distant vision, neither are they needed for reading or close work until that age is reached when the accommodative power begins to decline—*i. e.*, about the forty-fifth year. No great departure from emmetropia can long exist without producing more or less disturbance of the function of vision and of the nutrition of the ocular tissues, and without originating the numerous and complex general and reflex symptoms which arise from "eye-strain." To restore the eye, the refraction of which is abnormal, to a condition of emmetropia, or at least one approaching it, constitutes a most important part of the practice of ophthalmology. This will be more readily conceded when it is remembered that emmetropia is comparatively uncommon, occurring in not more than 1.5 to 2 per cent. of properly examined eyes.

Ametropia.—To the eye which fails in the requirements just described the term *ametropia* is applied, and the condition may be defined as follows:

Ametropia is any departure from the normal optical condition—that is, from an exact correspondence between the visual axis and the focal length of the dioptric apparatus when at rest. The principal focus is not a point or does not lie on the retina.

Ametropia is denominated *axial* when the length of the eyeball is increased or diminished, and *curvature* when, the axis remaining unchanged, the curvature of lenses of the eye undergoes variations. Ametropia presents itself under three conditions: (1) *Hypermetropia, hyperopia, far-sightedness,* or *oversightedness;* (2) *myopia, short-sightedness,* or *near-sightedness;* (3) *astigmatism* or *astigmia.*

It is convenient to distinguish the first two classes of ametropia by the relative position of the principal focus to the retina.

Hyperopia *is that form of ametropia in which the retina is situated in front of the principal focus of the eye. The visual axis of the eye is shorter than the focal length of the dioptric apparatus when at rest.*

The far point of the eye is negative, and is represented by the point behind the eye to which rays must converge before entering the eye, in order to be united on the retina. The refractive apparatus of the hyperopic eye, in a condition of minimum refraction, is adapted to bring rays converging to this point to a focus on its retina. Rays passing out of a hyperopic eye have a divergence as if they came from this point.

Causes and Varieties.—The eyeball may be abnormally short, constituting *axial hyperopia;* a deficiency of 1 mm. in the length of the optic axis produces 3 diopters of hyperopia; or its refractive power may be deficient, *curvature hyperopia;* an increase of 1 mm. in the length of the radius of curvature of the cornea produces a hyperopia of 6 diopters; or the crystalline lens may be absent, *aphakial hyperopia.*

Hyperopia is further divided into: (1) *Manifest;* (2) *latent;* (3) *total.* Manifest hyperopia (H. m.) is represented by the strongest convex lens through which an eye with perfectly intact accommodative power retains distinct distant vision; latent hyperopia (H. l.) is the amount in excess of the manifest which can be developed by the use of a cycloplegic—for example, atropin; total hyperopia (H. t.) is the sum of the manifest and the latent—that is, the entire amount of the hyperopia which is developed after paralysis of accommodation or complete relaxation of the ciliary muscle. Evidently latent hyperopia is the difference between the manifest and the total. Manifest hyperopia is either *facultative* or *absolute*—facultative when it can be overcome by an effort of accommodation, absolute when it cannot be overcome by an effort of accommodation.

Hyperopia is nearly always *congenital,* and is often *hereditary,* especially its high grades. In some senses it may be regarded as due to an imperfect development of the eyeball, which, however, may increase its length with the growth of the rest of the body, and this refractive condition may diminish, pass into emmetropia, or, more rarely, into myopia.

An apparent increase of hyperopia due to failure of accommodation caused by advancing years is often seen—that is, latent hyperopia becomes manifest. A real tendency to slow increase of hyperopia is due to gradual increase of the size of the crystalline lens. In early life none of the existing hyperopia is absolute unless it is of high degree; after sixty-five practically all of it becomes absolute.

Symptoms.—Hyperopia renders it difficult to maintain a distinct image of small objects—*e. g.*, printed matter—for prolonged periods of time. If the effort is persisted in, the accommodation becomes exhausted, aching of the eyes and head—in short, the result of *eye-strain*—appears, and finally the work must be discontinued (*accommodative asthenopia*). Sudden failure of accommodation, with consequent blurring of vision, is frequent, and often first appears if the patient has been weakened by illness. Hyperopes often place a book or small objects in a strong light in order to contract the pupil and thus render vision clearer.

Hyperopia frequently gives rise to *spasm of the accommodation*, owing to the persistent contraction of the ciliary muscle necessary to overcome this error of refraction, and then simulates myopia, distant vision becoming indistinct. In these circumstances concave lenses may improve vision, and, in ignorance of the true state of affairs, are sometimes prescribed, much to the detriment of the patient. A mydriatic will reveal the real condition of the refraction. It occurs also in myopic eyes, which appear to be more myopic than they really are. Spasm is prone to occur in individuals of neurasthenic condition, and is a frequent symptom of hysteria, often associated with cramp of convergence; it bears no relation to the vigor of the accommodation, inasmuch as persons with relatively feeble accommodation may have a marked cramp of the ciliary muscle. Therefore spasm of accommodation is not excess of accommodation (see page 41). Spasm of accommodation is not confined to young subjects, but may occur, and in stubborn form, after the fortieth year of life. It may be produced in normal eyes by the instillation of a solution of an active miotic.

Insufficient power of accommodation, *i. e.*, *subnormal accommodation*, may give rise to marked asthenopia. It is attributed by Theobald to a congenital insufficiency of the ciliary muscle (see also page 41), and its determination and relief in refractive work, as he has insisted, is most important.

Convergent strabismus is often the earliest symptom of hyperopia in childhood; it arises in connection with efforts of accommodation. When the hyperopia is too great to be managed by the accommodation, the affected children frequently hold their books close to their eyes, and, by contracting the palpebral fissures, are enabled to see better than with the book at a greater distance, because the object is seen under a larger visual angle, and the narrow slit between the lids cuts off the more divergent rays. These children are often erroneously supposed to be near-sighted, and concave glasses are given to them, which increase, instead of mitigating, the trouble.

As a result of hyperopia the coats of the eye become inflamed.

Conjunctivitis, blepharitis, styes, chalazia, and congestion of the retina and choroid are very frequent complications. *Persistent headache*, aggravated by using the eyes, various nervous symptoms, reflex in their nature, as well as disturbances in the visual function, are the common results of hyperopia (see also page 141).

Determination of Hyperopia.—Hyperopia always exists: When distant vision is not made worse by a convex glass; when the patient can read fine print through a convex glass at a greater distance than its focal length; when with the ophthalmoscope the interior of the eye, otherwise normal, is seen distinctly with a convex lens; usually when the near point lies at a greater distance from the eye than is proper for the age; and when the phenomena described in connection with the shadow-test on page 114 are present. To ascertain the presence of latent hyperopia a mydriatic should be employed. Its use is imperative in the presence of spasm of accommodation. The ciliary muscle is fully developed in hyperopic eyes, especially the circular fibers, which may be overdeveloped.

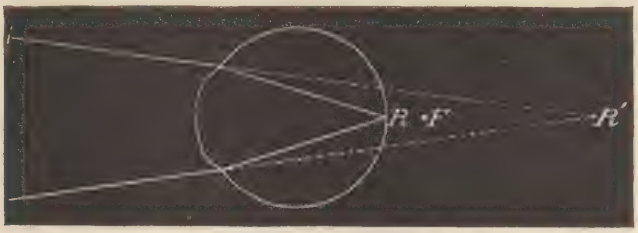


Fig. 61.—Far point of a hyperopic eye. Rays from R on the retina of the hyperopic eye after refraction diverge; these rays, prolonged backward, would unite at the point R'. R' is the far point.

Correction of Hyperopia.—The principal focus, F, of the hyperopic eye lies behind the retina. Consequently the retina, R, is situated within the principal focus, and its conjugate focus or far point, R', is virtual (Fig. 61). Rays from R seem, after refraction by the eye, to have come from R'; conversely, rays converging to R', after refraction by the eye, unite in R on the retina. The rays which come from the retina, R, of such an eye, after emerging from the eye are divergent, and, prolonged backward, would unite in the point R'. The distance of this point from the cornea is the focal length of the glass which corrects the hyperopia. The amount of divergence of the emergent rays is dependent on the degree of the hyperopia—that is, the distance R lies in front of F. The higher the degree of hyperopia is, the farther R lies in front of F, and the nearer the point of divergence R' lies to R; conversely, the lower the degree of hyperopia is, the nearer the point R lies to F, and the farther back the point R' lies. The distance of R' must be less than infinity; otherwise, the eye would be emmetropic.

If parallel rays are given a convergence to the point R' by a convex lens placed before the eye, the rays will come to a focus at the point R on the retina, since the path of the rays passing into the eye after refrac-

tion by a convex lens (Fig. 62) is exactly the same as that of the rays diverging from the retina and passing outward (see Fig. 61), only the direction is reversed. The far point, R', of the hyperopic eye is the point to which parallel rays must be given a convergence by a convex lens in order to come to a focus on the retina. The amount of this necessary convergence represents the deficiency between the refraction of the hyperopic and that of the emmetropic eye; the degree of hyperopia is, therefore, in an inverse ratio to the distance of R'.

To correct hyperopia the refraction of the eye must be increased by a convex lens of sufficient strength to bring F on the retina. This glass corrects the hyperopia by shortening the focal length of the dioptric apparatus to correspond exactly with the length of the visual axis. The far point R' is removed to infinity. Parallel rays come to a focus on the retina without any effort of accommodation, and rays emerging from the eye are rendered parallel.

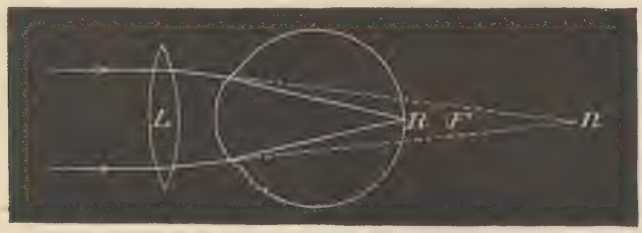


Fig. 62.—Correction of hyperopia by a convex glass. The lens, L, gives to parallel rays a convergence toward the point R'; they will consequently be united on the retina, R. R' is the virtual conjugate focus of R.

In order to neutralize the hyperopia that convex glass must be selected which gives the greatest visual acuteness. As this is obtained when the retinal image is sharply formed, and as this occurs when rays are brought to an exact focus on the layer of rods and cones, the maximum sharpness of sight is the most satisfactory evidence that rays are exactly focused on the retina. If these rays are parallel, the glass which brings them to a focus on the retina corrects the hyperopia. Rays from objects at 6 meters' distance are sufficiently parallel for this purpose.

Correction of Hyperopia with Test-types and Trial-lenses.—The card of test-letters, in good illumination,—either artificial light or ample daylight,—is hung on a wall, at 4 to 6 meters from the patient. A pair of trial-frames is placed before the patient's eyes and one eye at a time examined, the other being screened by an opaque disk. The patient is supposed to have his accommodation paralyzed by a mydriatic, or to be beyond fifty years of age. He is required to read the smallest letters which he can see distinctly on the card. The resulting sharpness of vision is noted. A convex glass is now placed before the eye. If this glass improves vision, but does not raise it to normal, stronger lenses are tried until the one is obtained which yields the maximum visual

acuteness; or, if the stronger glasses do not improve the vision, successively weaker ones are tried until that glass is found which gives the greatest sharpness of sight. This is the lens which corrects the hyperopia. If the acuteness of vision is raised to normal by a convex spheric lens, it is not likely that astigmatism is present, but every eye should be examined with a view to discover any astigmatism. If none exists, the convex glass is all that is required to correct the ametropia.

In the absence of a mydriatic and the presence of some accommodative spasm, vision being equal in the two eyes, a more suitable glass may often be obtained by testing both eyes simultaneously, because with parallel axes the accommodation is more likely to undergo relaxation. This effect may be further increased by placing a prism of 2° or 3° (centrads) before one eye, with its base inward. The effect of this is to relax the internal recti muscles, and indirectly the accommodation. It is a good plan to begin by placing before the eyes a lens of stronger refraction than the one required, and gradually weakening it by concave glasses of successively higher numbers until normal vision is reached. The glass required is then the difference between the two.

The proof that the glass selected is the correct one depends upon the ability of the patient to focus parallel rays on the retina. Parallel rays may be obtained by placing an object at the principal focal distance of a convex lens. The principal focal distance of a 4 D lens is 25 cm. Therefore if the glass corrects the hyperopia, the patient should be able to read fine print at 25 cm. distance with + 4 D added to his correction. If he reads at a greater distance than 25 cm., some hyperopia is still uncorrected. If he reads at a shorter distance than 25 cm., the hyperopia is probably overcorrected.

The degree of hyperopia may also be determined by placing a convex lens before an eye the accommodation of which is paralyzed, and by finding the distance at which small type appears most distinct. Suppose the lens selected is 4 D (focal distance = 25 cm.), and that the patient reads best at 33 cm. Now 33 cm. is farther than the principal focus, and the rays, therefore, are convergent after passing through the lens, since a 3 D lens would render them parallel; 4 D = 3 + 1 would give them a convergence of 1 D to the conjugate focus, 1 meter back of the eye; 1 D, therefore, represents the amount of the hyperopia (see page 126).

Rule.—Subtract from the lens employed the lens whose focal distance equals the distance at which the patient reads. The difference is the degree of hyperopia.

Correction of Hyperopia with the Ophthalmoscope and Shadow-test.—To correct hyperopia in children before they are old enough to read, the ophthalmoscope and skiascopy are the means upon which reliance is placed, but these methods should also be employed in adults. They are explained on pages 106 and 111.

Ordering of Glasses.—After the degree of the hyperopia has been determined, the very important question presents itself, What glass shall be ordered? While the eye is under the influence of the cycloplegic,

distant vision is distinct with the full correction; after the effects of the drug have disappeared, it is often dim with the full correction, and a haze seems to lie over all distant objects, which disappears when the glasses are removed. On the other hand, the headache, asthenopia, and congestive troubles return if the hyperopia remains uncorrected. Spasm of accommodation is the disturbing factor in this problem, and it is so variable in different individuals that no precise rule can be given. Many persons wear a full correction with comfort, and do not need any modification; others will tolerate only a small part of the full correcting glass.

There are two methods of dealing with this difficulty: first, to order full correction while the eye is still under the influence of the mydriatic, and to insist that this shall be worn constantly during the time that the accommodation is returning to its normal state. If distant vision remains dim, after full accommodative power has returned, the glasses may be weakened sufficiently to secure normal sight for long ranges.

It should be borne in mind that the glass which gives the best correction at 4 or 6 meters is not the correcting glass for the total H, but, in reality, is an overcorrection of $\frac{1}{4}$ to $\frac{1}{6}$ D. Strictly speaking, rays coming from these distances are not parallel, and the glass which focuses them perfectly on the retina will not perfectly focus parallel rays. Hence, in ordering a full correction, the glass which gives the best vision at 4 or 6 meters must be weakened by $\frac{1}{4}$ or $\frac{1}{6}$ D. If this fact were more often remembered, less difficulty would be experienced in inducing patients to wear a full correction.

Second, the eyes are first allowed to regain their full power of accommodation before the final glass is prescribed, and this is the plan which should be pursued. If vision is normal with the full strength of the glass, it may be ordered; if not, it should be reduced to that number with which full visual acuteness is obtained. This may be only one-half, one-fourth, or even less, of the full amount. It is necessary in these cases to increase the strength of the glass from time to time as symptoms of fatigue manifest themselves. When the glass ordered for distance is only a small part of the full correction, another pair of lenses for reading may be ordered which embodies nearly or quite the full amount of correction.

A frequent cause of inability to wear a full correction depends upon the development of convergence insufficiency, causing an associated action of accommodation with the muscular effort necessary to bring the visual axes into a parallel condition (see page 556).

Instead of ordering the glass nearest in strength to the full correction, with which the patient still has normal vision, the lens which neutralizes the total hyperopia may be reduced by a given amount, usually 0.75 D. Donders advised a glass based upon the manifest H, to which one-quarter of the latent H was added. Macnamara recommends, in absolute hyperopia, the use of a convex glass, the strength of which shall be equal to one-half of the sum of the manifest and total hyperopia—*e. g.*, manifest H = 1.5 D; total H = 3.5 D. H. m. + H. t.

= 5 D; ordered + 2.5 D. The author, if convergence is ample, usually orders the full correction of H less 0.25 D. If there is exophoria, this plan must be modified, or the defect remedied by prisms or by prismatic exercises. The indistinct vision, caused by full correction of H, due to a disturbance of the relative range of accommodation and convergence, may be overcome by systematically training the convergence (see page 559). Whether a glass shall be worn constantly or not depends upon the symptoms which the hyperopia has produced and the character of the patient's work. Frequently hyperopes are entirely comfortable if reading-glasses alone are used. Finally, glasses need not be ordered simply because hyperopia exists; but only when it gives rise to the symptoms which have been described. Thus, one person may easily manage one or more diopters of H without glasses; another may have all manner of asthenopic and reflex nervous symptoms produced by 1 D of H or even less.

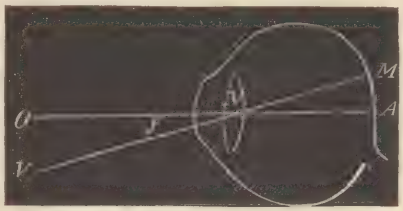


Fig. 63.—Angle gamma in hyperopia. *O–A*, The optic axis; *N*, the nodal point of lens; *V–M*, the visual line, cuts the cornea at inner side of optic axis; *O–N–V*, the angle gamma, in this case is positive; *M*, the macula.

The visual line is often very much displaced to the inner side of the cornea in hyperopia, causing a very large value of the angle gamma.

Myopia *is that form of ametropia in which the retina is situated behind the principal focus of the eye, and only those rays which diverge from some point nearer than infinity can come to a focus on the retina. This point is the far point of the myopic eye.*

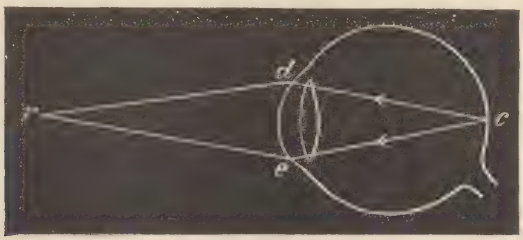


Fig. 64.—Far point of a myopic eye. Rays diverging from the retina, *c*, will, after refraction, converge to *r;* conversely, rays diverging from *r* will, after refraction, converge to *c; r* is the far point; *r* and *c* are also conjugate foci.

The far point, therefore, is limited by the amount of divergence necessary to bring the focus of the rays on the retina. The higher the degree of myopia is, the closer will the far point *r* lie to the eye. Rays coming from the retina converge to the far point and form there an image (Fig. 64). This image can be seen by the ophthalmoscope. The far point and the retina are conjugate foci (see page 113).

Cause and Varieties.—Myopia may be produced by increased refraction of the cornea or crystalline lens, *curvature myopia,* or by too

great a length of the optic axis, *axial myopia*. In the majority of cases myopia is due to elongation of the optic axis, often the result of pathologic changes in the coats of the eye.

Myopia may also be occasioned by changes in the shape of the cornea as a result of disease—for example, conical cornea. *Corneal opacities* are a frequent cause of myopia. According to Frenkel, bilateral opacities usually produce bilateral myopia, while unilateral opacities more frequently give rise to unilateral myopia, which may affect either eye, according as the one or other eye is most used for near vision. The myopia thus caused appears to be an axial and not a curvature myopia. Myopia, unlike hyperopia, is rarely congenital. It usually makes its appearance from the eighth to the tenth year, and tends to be progressive, especially during the early school years. Sometimes it is the continuation of a process started in hyperopic eyes, especially in those with astigmatism, and the gradual transition from hyperopia to myopia is not infrequently seen among patients who return for examination. According to Risley, there may be an arrest of the increase of myopia as the result of treatment and the optical correction of ametropia.

Myopia is more prevalent in some countries than in others, and is especially frequent in Germany, in the higher classes of the schools, reaching, according to Cohn, 60 per cent. Myopia is said to be more common among Jews than among Christians of the same social class (Sydney Stephenson). Sattler, however, is unconvinced that there really exists a racial inclination to myopia. Harman's statistics indicate that most of the cases of myopia in early childhood occur in boys, but the highest degrees appear in girls. Although the largest number of myopes is found among the upper classes,—that is, among those upon whom the demands of modern civilization fall most heavily, and among artisans whose work requires close inspection,—high grades of this refractive defect may also be found among those who apparently do not use their eyes for close work, and occasionally among children who have not yet been subjected to the influence of school life. It may be that it will be found that these subjects of myopia have been reared under conditions in which they have been obliged to devote themselves assiduously to work at very near range (Sattler).

Myopia is frequently hereditary, and may occur in several members of one family. Harman found hereditary transmission in 9 per cent. of the cases he investigated. With a strong predisposition to myopia the elongation of the eyeball may take place under comparatively unimportant exertion. Although myopia usually begins in childhood, there may be a *late development* of this refractive error, that is, it may occur after the twentieth year of life. This late development of myopia has been observed in association with constitutional disturbances, with malaria (T. Becker), with goiter and obesity (R. Wirtz), and with pituitary body struma.

That acute posterior scleroticochoroiditis may occasion myopia, especially posterior staphyloma, as originally taught by Von Graefe, is

not admitted by Sattler, that is, that it is the cause of the myopia. Obvious choroiditic changes, according to him, are to be considered as a complication which etiologically has nothing to do with the progression of the myopia; although he admits that choroiditis may give rise to a rapid increase and pernicious course in myopia. The author has published some observations which indicate that, as the result of severe choroiditis, myopia may rapidly develop in certain cases, observations which are in accord with the statements of Knies, Priestley Smith, and others. Occasionally after an acute illness, especially one of the exanthemata, a myopia develops, or there may be a sudden increase in a pre-existing myopia. Alteration of the refractive power of the lens as the result of beginning cataract may cause myopia, the so-called *second sight* (see page 395), and, according to Hirschberg, the late development of myopia—that is, after the fortieth year, unassociated with cataract formation—is not an uncommon sign of diabetes. *Transitory* increase of *hyperopia* has also been observed in diabetes, due to changes in the lens (Lundsgaard).

In normal eyes the sclera does not yield to the intra-ocular pressure, but if from any cause its resisting power is reduced, distention takes place and the anteroposterior axis of the eyeball is elongated. Among the causes which have been invoked to explain the elongation of this axis of the eye—*i. e.*, the production of myopia—are the following: The incentive given by the shape and size of the orbit to greater development of the eyeball; the compression of the eyeball by the external muscles, causing distention of its coats backward on account of the excessive convergence rendered necessary by the close range at which myopes are obliged to work;[1] the strain of accommodation; racial peculiarities; inflammatory changes within the eye—for example, sclerotico-choroiditis, induced by habits of life which promote fulness of the veins of the head and neck and hinder the egress of the blood from the eye or are set up by eye-strain itself induced by excessive study, bad ocular hygiene, imperfect illumination, etc.; and an inherited tendency, the commencement and increase of the myopia being caused by general and local vascular congestion, which are the result of constitutional disturbance—for example, cardiovascular disease (Batten).

Although prolonged use of the eyes at near work necessitating excessive convergence and muscle pressure explains the acquisition of myopia in many cases, only a portion of those subjected to such a strain become myopic. Therefore in this number, as Fuchs remarks, special additional factors must be present: predisposition, too great approximation of the work, improper ocular and general hygiene, spasm of accommodation, and, especially, astigmatism. Irregular, inverse, and oblique astigmatism are, according to Duane, of marked significance in this respect.

[1] Compression of the eyeball under these circumstances may be caused by the external rectus. According to Stilling the superior oblique is the principal compressing muscle in myopia, the low position of the trochlea increasing the amount of force which this muscle exercises on the globe. Schmidt-Rimpler rejects Stilling's conclusions, and Hamberger's measurements do not confirm Stilling's contention that in myopia the vertical diameter of the orbit is decreased.

Among other causes of less moment may be mentioned an unusually great distance between the pupils, rendering convergence more difficult, a divergent squint, and a large size of the angle gamma (in this case negative), demanding more strain on the part of the eye muscles in the efforts of convergence. After myopia is once produced the eyeball, by its oval shape and greater size, may act as a cause of the further development of this refractive defect by reason of the increased muscular effort which is required to rotate such a globe inward during convergence, and the compressing effect of the external recti muscles on the increased posterior segment of the eyeball.

At first probably all cases of myopia are *progressive*, but many are checked because the eyes are removed from the strain of close work or are placed under better hygienic surroundings; that is, the myopia becomes *stationary*. Other cases may progress until the increased effort of convergence demanded by the increased myopia becomes too difficult to sustain, one eye deviates outward and there is produced a *divergent strabismus*. Then further increase of myopia may stop, or the inflammatory changes already set up within the eye may continue, the distention of the ocular coats increases, and the most serious organic lesions arise; in other words, there is *malignant* or *pernicious myopia*.

Symptoms.—The symptoms of myopia naturally range themselves under the two classes, subjective and objective.

The *subjective symptoms* are those which arise because the range of vision is limited by a radius of a few centimeters. Distant objects are not clearly perceived by the myopic patient, because as soon as an object passes beyond his far point it becomes indistinct. According to Seggel, the *light-sense* diminishes with an increase of myopia. Percival Hay finds that if refractive errors are low, they do not affect the light-sense, but if they are high they tend to increase the light difference.

Many myopes have an inclination to avoid outdoor sports on account of their poor vision, and exhibit a greater fondness for occupations which come within their range—*e. g.*, reading, drawing, etc.—than for others which require good distant vision. The prolonged congestion of the eyes which such habits entail tends to increase the myopia. Headache and reflex phenomena are unusual accompaniments of myopia unless complicated with *astigmatism*, which is an important factor in the further increase of the refraction. Myopia, however, frequently causes aching of the eyeballs, very imperfect ocular endurance, congestion of the conjunctiva—indeed, many of the symptoms which are strictly asthenopic, especially when the choroid is undergoing the changes which are determining the increase in the refractive power. That the full enjoyment of outdoor sports is not at all incompatible with the existence of myopia properly corrected is well known, and it is an interesting fact, pointed out by Goldberg, that many excellent marksmen are myopic.

The *objective symptoms* of high myopia may embrace: (1) A notably prominent, and elongated eyeball, with a large and somewhat sluggish pupil; (2) a rather stupid expression of the countenance from inability

to note the expression in the face of others; (3) a peculiar manner of reading—the book is held stationary and the face is moved from side to side, following each line; (4) certain characteristic ophthalmoscopic appearances. With the direct method the optic disk appears enlarged; at its outer side there often is a crescentic area of whitish hue, depending upon alterations in the choroid, known as a *conus* or *myopic crescent.* This area may begin next to the disk with a space of complete atrophy, succeeded by a rim of partial atrophy and pigment disturbance, which in its turn merges into a patch of choroidal congestion. Sometimes the entire disk is surrounded by areas of choroidal disturbance, and the general choroid may exhibit many alterations depending on congestion, edema, rarefaction, atrophy, and pigment accumulation (see also page 130 and Fig. 65). Weiss and B. Alex. Randall have described a curvi-

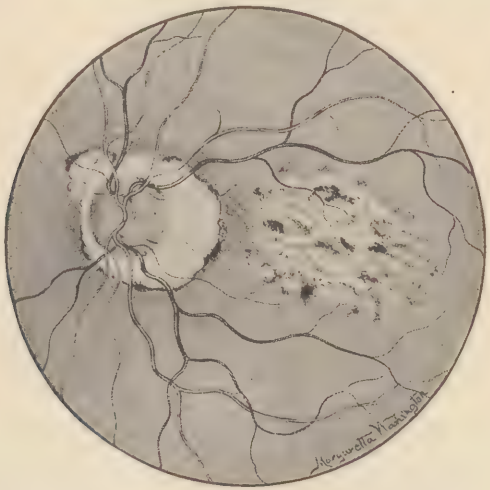


Fig. 65.—Eye-ground in progressive myopia. Large posterior staphyloma surrounding the nerve-head. Macular region occupied by areas of semi-atrophic retino-choroidal lesions.

linear reflex, generally at the nasal side of the disk, as a prodromal sign of myopia. (5) Divergent squint. The squinting eye is often amblyopic. Binocular vision does not exist in such a case; the better eye, freed from the necessity of convergence, reads at the far point without any effort, and glasses for reading are sometimes unsatisfactory because the print appears smaller on account of its removal to a distance greater than the far point of the eye.

The visual axis in myopia sometimes passes through the cornea at the outer side of the optic axis; the *angle gamma* is then *negative,* and the eye in looking at a distant object turns inward in order to bring the visual line to fix on it, giving rise to an apparent convergent squint (Fig. 66). This renders necessary a greater degree of convergence.

Myopic eyes are popularly considered as strong eyes because they see fine print at close ranges. This is true only in those cases in which

the tunics of the eye have suffered no injury—where, for example, the myopia is of moderate degree and the eye-grounds are normal.

Myopia does not usually decrease with age, but, on the contrary, tends to increase up to adult life or later.

Very high (10 D and higher) degrees of myopia (*malignant* or *pernicious myopia*) are often marked by ravages in the structure of the choroid and retina. The pigment-cells wander off in some places and accumulate in others, producing marked contrasts in the appearance of the eye-ground. Large areas of atrophy, glistening white in color, alternate with black splotches, and at times hemorrhages occur. The macular region is especially prone to degenerative, atrophic, and hemorrhagic changes. According to Hirschberg, these central changes, characteristic of high myopia, are due to mechanical stretching and not to inflammatory processes. The disk is often surrounded by an atrophic area, the *posterior staphyloma,* which represents an area of thinned and distended sclera.

Posterior staphyloma should not be confused with the conus or myopic crescent, and, according to Schnabel, who accepts von Jaeger's view, it should be regarded as an anomaly in the form of the eye—that

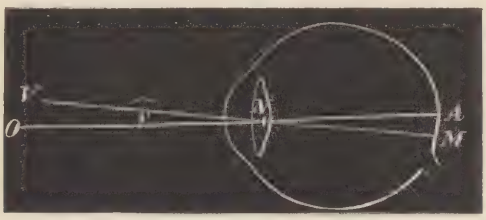


Fig. 66.—Angle gamma in myopia which is negative.

is, a malformation and not the result of disease. He believes that there is a connection between retinochoroiditis and posterior staphyloma, but that "the primarily emmetropic or hyperopic eye will not become affected with retinochoroiditis of the macula in consequence of acquired myopia; only eyes with posterior staphyloma resulting from congenital malformation have, in addition to excessive myopia, an especial predisposition to that grave disorder."[1] The vitreous humor is semifluid, and floating opacities are often visible, sometimes being so large as to obscure vision. Owing to the intimate relation between retinal nutrition and the pigmented epithelium of the retina, the loss of the latter is followed by diminution in the visual acuteness. In high grades of myopia—15 to 20 D, and sometimes still higher—the condition of the eye is desperate, and the morbid processes may culminate in detachment of the retina and complete blindness. Occasionally, in the macular region of myopic eyes may be seen an intensely black area, about the size of the nerve-head, with a slightly grayish center, and surrounded by a lighter ring. It stands out well

[1] "Relationship of Staphyloma Posticum to Myopia," I. Schnabel, System of Diseases of the Eye, edited by Norris and Oliver, vol. iii.

defined. This is the so-called *black spot of the macula in myopia*, first described by Förster and later by Fuchs. It is interpreted in the visual field by a scotoma. The prognosis is unfavorable and the disease may be progressive, although, according to some observers, this lesion renders the eye less liable to the other complications of myopia. Butler and Stargardt have described a central *green spot* in myopia.

Fig. 67.—Ciliary body of a myopic eye (specimen prepared by Dr. C. M. Hosmer). Notice the abnormally flat appearance of the ciliary body.

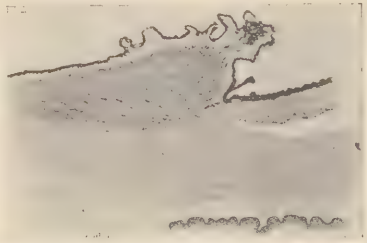


Fig. 68.—Ciliary body of a hyperopic eye (speciman prepared by Dr. C. M. Hosmer).

Not only is corneal astigmatism a potent factor in the increase of myopia, but, according to Senn, it bears an important relation to central choroiditis and destructive change in the fundus.

In myopia the ciliary body appears to be flat, and the transverse diameter of the ciliary muscle is smaller than it is in the normal eye, because its circular fibers, comparatively little employed in the act of accommodation, are poorly developed. The sinus of the anterior chamber is deeper than it is in emmetropic or hyperopic eyes, and

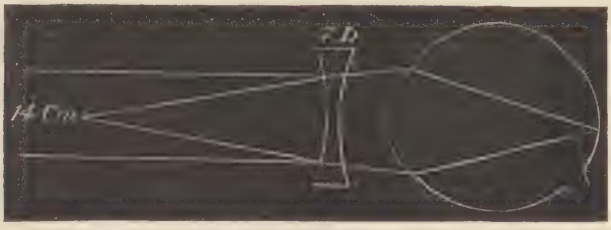


Fig. 69.—Manner in which a concave lens causes rays to diverge from the far point of a myopic eye.

hence the tendency to primary glaucoma in myopic eyes is said to be lessened.

Determination and Correction of Myopia.—Myopia may be determined: (1) By the position of the *punctum proximum* of accommodation, which is closer to the eye than is normal for the age; (2) by the position of the farthest point of distinct vision obtained by test-types; (3) by the ophthalmoscope and retinoscope (page 113); (4) by the concave glass which gives distinct vision at a distance of 4 to 6 meters.

Only those rays which diverge from a distance not greater than the

far point can be focused on the retina of the myopic eye. In order that it shall see at any greater distance than this the rays must be given a divergence as great as if they came from this point (Fig. 69). If the greatest distance at which a myopic eye can see fine print is 14 cm., in order to see at a still greater distance the eye would require a concave glass which would give rays a divergence as if they came from this point. By dividing 100 by 14 we obtain the number of diopters (7) necessary to produce this divergence. As the far point is measured from the cornea, the glass must be placed close to the cornea; if the glass is removed 1 cm. from the cornea, it is plain that its focal point will also be 1 cm. farther away; therefore it is necessary to employ a glass of shorter focus.

Example.—Suppose it is desired to cause the rays to diverge from a point 14 cm. in front of the cornea, and the glass is to be placed at 1.5 cm. in front of the cornea; it is evident, under these circumstances, that the glass would require to have a focus of $14 - 1.50 = 12.5$ cm., or $\frac{100}{12.5} = 8$ diopters.

The usual position for a glass is about 13 mm. in front of the cornea.

In low degrees of myopia this does not affect appreciably the strength of the glass, but in the higher degrees it makes a serious difference. The concave glass is, therefore, somewhat stronger than the actual myopia, especially in the higher grades.

The degree of myopia may be determined approximately by this method more rapidly than by beginning the trial at 6 meters with glasses (in this instance, concave) in the manner already described in connection with hyperopia (see page 124). One example will suffice:

A patient reads fine print distinctly at 8 cm. from the cornea, but not at a greater distance, the eye being under the influence of a cycloplegic; this is its far point. In order that the patient may see at an infinite distance, parallel rays must be given a divergence as if they came from 8 cm. in front of the cornea. As the glass will be placed 13 mm. in front of the cornea, its focal length must be 8 cm. $-$ 1.3 cm. $=$ 6.7 cm., or 67 mm. $\frac{1000}{67}$ mm. equals 15 D, as the number of the concave lens required to permit distant vision. A lens of this number should be placed in the trial-frame, and the vision determined through it by means of test-types at the usual distance. Perhaps a weaker or a stronger lens may give better vision, and hence several numbers should be tried in succession, until that glass is selected with which the greatest acuteness of vision is attained, and which then represents the correcting lens.

A patient often will select a glass of higher number than the one really required, because the letters have a blacker and sharper appearance when seen through concave lenses; but unless the stronger glass at the same time secures for the patient an increased acuteness of vision it should be rejected, and the weaker lens adopted. If several lenses give equally good vision, the weakest one should be chosen.

The method of determining the correcting lens in myopia by means of ophthalmoscopy and skiascopy is elsewhere described (see pages 106 and 111).

Treatment of Myopia.—This should include *prophylactic measures* and the selection of suitable concave glasses. From the eighth to the eighteenth year—that is, during school life—myopia specially tends to appear and to progress; hence prophylactic means are urgently required during this period. No child should be permitted to begin school duties until the exact state of the refraction has been determined, and if vision is deficient and eye-strain likely to develop, suitable lenses should be prescribed. Strict attention should be paid to the following conditions: A correct position of the head and body during study, secured by means of a suitable desk, the surface of which is so tilted that the page of the book lying on it is parallel to the scholar's face, and by a chair or stool of proper height both in relation to the desk and the floor; the employment of books with sufficiently large and distinctly printed type; good illumination coming from behind the scholar and preferably over the left shoulder; proper ventilation; restriction of the hours of study within reasonable limits and plenty of outdoor exercise. These precautions apply with equal force to hours of study at home.

As Priestley Smith has well said, it is necessary "to suspect every myopia, and especially every youthful myopia, of a tendency to increase, until time has proved it to be stationary; to be doubly suspicious in the presence of congestion or atrophy of the eye-ground; and to reexamine at intervals of six months, twelve months, or longer, according to the nature of the case." These examinations should be made with the help of mydriasis—if possible, with atropin. It is particularly important frequently to investigate the eyes of the children of myopic parents. In some cities—for example, in Berlin—a special curriculum is provided for children with a myopia of 6 D and over (Hirschberg). Harman also urges that children with high degrees of myopia should be instructed in special classes.

If a tendency to divergence exists in early life, it is sometimes proper to remove this by tenotomy of the external rectus as a preventive measure against the development of myopia.

Since Fukala's recommendation removal of the crystalline lens (discission, followed by extraction, or *phakolysis*) has been practised by a number of operators for the relief of high myopia (15 D or more). Improvement in vision and increase in the distance at which eyes can be used in near work are the results of successful operations, which, according to von Hippel, may not reach their best standard until a year after the operation—checking of the increase of the myopia.

The chief dangers of the operation are: Intra-ocular hemorrhage, detachment of the retina, secondary glaucoma from swelling of the lens, iritis, and infection of the corneal wound. The chief contraindications are: Extensive degeneration of the choroid, retina, or vitreous, diminished intra-ocular tension, a tendency to intra-ocular hemorrhage, previous loss of one eye from any cause, and advanced age. Hirschberg vigorously condemns any tendency to indiscriminate application of the operation and has operated only on carefully selected eyes. (For methods of operating see pages 649, 650.) The author's experience

with the operation has been very limited, but it has been favorable. He believes, however, that in the majority of cases correcting lenses, even in very high grades of myopia, can be made to serve a more useful purpose than operative interference, and has but rarely found it necessary to advise the operation. Hertel has designed *telescopic spectacles*. They somewhat resemble an automobile goggle. They decrease the size of the visual field, but increase the distinctness of distant objects. They are intended to be a substitute for operative procedures in high myopia.[1] To compute the probable correcting glass after loss of the crystalline lens, according to Landolt, one should divide by 2 the number of diopters of the correcting glass of the complete eye, and when concave, subtract it from 11 D, and when convex, add it to 11 D.

Ordering of Glasses.—After the estimation of the degree of myopia, astigmatism having been excluded, or, if present, corrected, the strength of the glass suitable for constant use, reading, or other special work must be determined. This is decided by the visual acuteness, the range of accommodation, the degree of the myopia, and the condition of the external ocular muscles.

Young people (under twenty) with good vision and a moderate degree of myopia (6 D and under) should wear the full correction constantly if the accommodation is ample and no signs of fatigue are evident.

Indeed, full correction is the object to be attained for young persons with normal visual acuteness and binocular near vision, no matter how high their myopia (Jackson), provided the lens selected shall not be an overcorrection when brought close to the eye. The author is convinced from personal experience that full correction, other things being equal, yields the best results in the management of myopia, and especially in the prevention of its increase, and this conviction is strengthened by the abundant statistical information on this subject which has been collected and analyzed by observers here and abroad. A few authors, for example, Hirschberg, are not in accord with this advice. Hirschberg advises that if the myopia is beyond 3 D, fully correcting glasses should not be worn. Naturally, there are exceptions to the rule, and each case requires thoughtful study; but, in general terms, the full correction of myopia yields the best results.

When visual acuteness is imperfect or binocular vision lost, it is usually better to order a partial correction for near work, and if the patient has attained those years when accommodation naturally fails, he must be provided with lenses for close range, or, if his myopia is of suitable degree, read without glasses. When wearing a partial correction the patient is tempted to improve distant vision by looking obliquely through the glass. But this gives it a cylindric effect, varying with the direction of the visual axis, and is always injurious.

In high grades of myopia associated with lowered vision it is often necessary to diminish the full correction from 1 to 3 D. It is evident that the greater the visual acuteness, the farther away the same size

[1] See Archiv. f. Ophthal., Bd. lxxv, Heft 3, p. 586, 1910.

of type can be seen; hence the demand on accommodation is less as the visual acuteness is greater.

When strong concave lenses are first worn, a lack of accommodation often appears, which is restored by a few months' use of the glasses. For the relief of this deficiency it is advisable to give a partial correction for near work until ample power of accommodation is gained, when the full correction may be used for all purposes.

As age advances an additional glass should be ordered for reading which will give the patient a far point of from 30 to 60 cm. In order to obtain this, the full correction must be diminished from 1.50 to 3 D.

The *position* of the lens used to correct high grades of myopia is of great importance. The nearer the lens is placed to the cornea, the stronger it becomes; conversely, the farther it is removed from the cornea, the weaker it is. The strong concave lenses necessary to correct high degrees of myopia in this way may sometimes be utilized by the patient to gain artificial accommodation. By bringing them close to the eye vision is adapted for distance; by pushing them from the eye divergence is lessened and the eye is adapted for a closer point.

The visual acuteness in high myopia is usually reduced, and in those cases accompanied by changes in the retina and choroid this reduction assumes a considerable grade. Sometimes very slight improvement in distant vision is secured by concave glasses, and near vision may not be at all benefited. In these circumstances patients see better by using one eye alone and bringing the print or other work close to the eye, because the enlarged retinal image compensates for the diminished visual acuteness. These cases, however, are seldom encountered, and a concave lens, properly selected, almost always improves both near and distant vision.

Concave glasses diminish the size of the retinal image, especially when the glass is removed farther from the eye. The retinal image is larger in myopia than in emmetropia, but if the correcting lens is exactly 13 mm. in front of the cornea, the image is of the same size as in emmetropia.

Concave lenses act as prisms when the visual line passes through any portion except the optical center. The optical centers should always be separated by a space equal to, and never less than, the interpupillary distance, except in those cases of weakness of the internal rectus muscles where it is advisable to increase the distance between the centers. This produces the effect of a prism with its base inward—that is, it lessens the amount of convergence which otherwise would be required. The deviation may be calculated from the focal distance of the lens and the amount of decentering. The distance the optical center is displaced, divided by the focus, equals the tangent of the angle of deviation. Myopes with decided esophoria often read more comfortably without than with glasses.

The painful glare of light sometimes caused by wearing concave glasses may be modified by tinting them.

The reading-glasses for myopes are described under Presbyopia.

Astigmatism (Astigmia).—In the preceding forms of ametropia, H. and M., the cornea has been considered as an ellipsoid of revolution, so that planes passing through it in various directions, vertical, horizontal, and oblique, produce sections having an equal curvature. Equal refraction, consequently, takes place in these different planes. Variations in the curvature of the different meridians produce differences in their refractive power; in some of these meridians the eye must, therefore, be ametropic. Three conditions may arise:

1. The eye may be emmetropic in one meridian and ametropic (either H. or M.) in the others.

2. The eye may be ametropic (H. or M.) in all meridians, but in different degrees.

3. The eye may be ametropic in all meridians, but in some H. and in others M. (H. and M.).

It is convenient to designate the different parts of the eye by imaginary lines, similar to those employed in geography.

The *axis* of the eye is a line drawn from the center of the cornea through the center of the ball. Passing through the center of the lens and the center of rotation, it penetrates the sclerotic between the optic nerve entrance and the macula. The anterior and posterior extremities of this line are the *poles* of the eye.

A great circle extending round the ball perpendicularly to the axis, and at an equal distance from the two poles is called the *equator* of the eye; other great circles passing through the poles are called *meridians*.

The lens is described in a similar way by its axis, anterior and posterior poles, and equator.

When the meridians of the cornea have an equal curvature, the rays of light gather in one common focus. Frequently the cornea has meridians of unequal curvature producing greater refraction in some meridians and less in others. The rays passing through the meridian of highest refraction reach their focus soonest, while those passing through the least refracting meridian come to a focus farther back.

Definition.—*Astigmatism (or astigmia) is an ametropia of curvature, and the term is applied to that refractive condition of the eye in which a luminous point—for example, a star—forms an image on the retina, the shape of which image is a line, an oval, or a circle, according to the situation of the retina, but never a point.*

Seat of Astigmatism.—Usually the cornea is the seat of astigmatism, but astigmatism may also be produced by an oblique position of the lens, or by the visual line passing eccentrically through the cornea.

When the meridians of the cornea progress evenly in their refraction from the lowest to the highest, the astigmatism is termed *regular*. When the curvature in different parts of the same meridian varies,—and the meridians vary irregularly in their curvature as the result of cicatrices from ulcers or distention of the cornea from inflammation,—the astigmatism is called *irregular*.

Almost all eyes possess more or less *irregular astigmatism*. Usually it is only slight, and gives no serious inconvenience for ordinary vision,

but all points of light, such as stars, distant street-lamps, etc., shoot out rays and twinkle as the result of the irregular astigmatism of the eye. The seat of this irregular astigmatism is in the crystalline lens. In the lenses of young people the union of the sectors is visible by three faint lines—the *lens star;* in the adult secondary rays are also visible. Slight differences in the density of the several sectors are sufficient to produce a distorted image of a luminous point.

Principal Meridians.—In regular astigmatism the cornea has one meridian with the shortest radius of curvature producing the highest refraction, and another meridian, at right angles to this, with the longest radius of curvature and the least refraction. These are called the *principal meridians*, and may be situated in any part of the cornea, but there is a disposition of the greatest refracting meridian to lie in or near a vertical direction, and of the least refracting meridian to lie in a horizontal direction.

If the meridian of greatest refraction is vertical or nearly so, the astigmatism is described as *with the rule;* when the meridian of greatest refraction is horizontal or nearly so, the astimgatism is spoken of as *contrary to the rule* or *inverse;* if the direction of the principal meridians approaches 45° and 135°, the astigmatism is often designated *oblique.*

To simplify the phenomena of astigmatism the principal meridians will be considered as running vertically and horizontally with the greatest refraction in the vertical, and the least refraction in the horizontal, meridian.

Form of the Image of a Point Focused by an Astigmatic Eye.—The rays passing into an astigmatic eye, thus considered, are most sharply refracted by the vertical meridian. The bundle of rays, instead of having a round section, forms a horizontal oval, which becomes smaller as the rays travel farther backward; but the vertical diameter of the oval lessens most rapidly until, when the focus of the vertical meridian is reached, the figure becomes a horizontal line, because all the rays are brought to one level and remain diffused only in the horizontal direction.

Farther back the rays, after passing this focus and crossing, diverge again vertically, and the figure becomes once more a horizontal oval; but shorter because the horitonzal diffusion is diminished.

Still farther the figure assumes the form of a circle; the diffusion of the horizontal rays has become less, and that of the vertical rays more. The figure becomes next a vertical oval, then a vertical line as the focus of the horizontal meridian is reached. Finally, the section is again a vertical oval, the horizontal rays, having passed their focus, cross and begin to diverge (Fig. 70).

It is evident from this that no matter what position the retina may occupy, no distinct image can be formed upon it, but there must always be overlapping of the images of the different points of an object, causing a blur or a wrong impression of its outline.

Symptoms.—In this manner the acuteness of vision is diminished

by astigmatism. Letters are not distinctly seen, some letters being confused with others—H and N, B and S, F and P, K and X, V and Y. The overlapping of the diffusion areas in the retinal image produces, in high degrees of astigmatism, an apparent doubling of the object. The indistinctness of vision compels a closer approximation of the object, with a consequent strain upon the accommodation.

Astigmatic persons learn to overcome their refractive defect by contracting the lids close together in order to make a horizontal slit. The

Fig. 70.—Retinal images of a point in the different forms of astigmatism: *A*, Compound hyperopic astigmatism; *B*, simple hyperopic astigmatism; *C*, *D*, *E*, mixed astigmatism; *F*, simple myopic astigmatism; *G*, compound myopic astigmatism.

vertically divergent rays are thus excluded, and the eye, accommodated for the horizontally divergent rays, receives a more distinct though fainter image. There is an almost characteristic facial expression in astigmatism caused by contraction of the lids.

Astigmatism produces an indistinctness in the appearance of fine lines running in certain directions, the direction of the indistinct lines being determined by that meridian which has its focus on or nearest

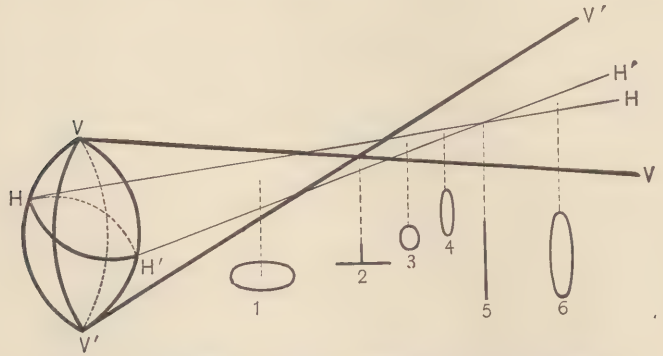


Fig. 71.—Rays passing through an astigmatic lens (Thorington).

to the retina. This meridian, therefore, will most nearly approach emmetropia; the lines parallel to it will appear indistinct, while those parallel to the opposite meridian, or the one farthest removed from emmetropia, are most distinctly seen.

In those cases in which the horizontal meridian is emmetropic and the vertical meridian ametropic, fine parallel lines running in a horizontal direction will appear spread out into thick bars, while vertical lines will appear distinct.

To understand this, the student should remember that rays diverge from a horizontal line in all directions; those which pass through the horizontal meridian, if they are not exactly focused, spread out in the direction of the line, causing its extremities to appear somewhat faint in outline, but do not blur its width. The rays which diverge in vertical planes from the different points in the line pass through the vertical meridian. If this is not emmetropic, the breadth of the line appears thicker; but if the vertical meridian is emmetropic, it forms a distinct point in the image, of each point in the object, by bringing the rays which pass through it to a focus. A horizontal line thus appears as a succession of distinct points when the vertical meridian is emmetropic. Vertical lines, in the same way, appear most distinct when the horizontal meridian is nearest to emmetropia, or if oblique lines appear most distinct, the meridian at right angles to their direction is the one nearest to emmetropia. Luminous points are drawn out in the direction of the ametropic meridian, and luminous circles become elongated into ovals.

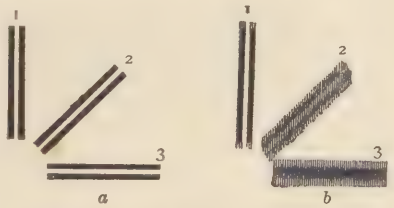


Fig. 72.—Illustrating the appearance of lines running in different directions as seen by (*a*) the normal eye and (*b*) the astigmatic eye (Jackson).

Astigmatism may be responsible for the most aggravated types of *asthenopia* and most marked symptoms of *eye-strain*. Fully 60 per cent. of functional *headaches* are caused by this type of refractive error, either alone or in association with other forms of ametropia. The headache may vary from a moderate frontal distress to violent explosions of pain, and may be situated in any portion of the cranium. That true *migraine* is caused by astigmatism alone is doubtful; that the correction of astigmatism is an important, indeed, an essential, part of the treatment of this affection should not be disputed. Furthermore, all manner of reflex nervous disturbances, vertigo, pseudochorea, habit-spasm, epileptiform convulsions, melancholia, neurasthenia, tachycardia, night-terrors, flatulent and other types of dyspepsia, indigestion, and even constipation are the frequent results of astigmatism, not only when the error is of high degree, but commonly, indeed, more commonly when it exists in low grade, and often unassociated with any symptoms which prominently direct attention to the eyes as the cause of the distress. Pains strangely and persistently situated in the nape of the neck, between and under the shoulder-blades, in the precordium, at the end of the spine, and deep in the mastoid may owe their origin to the same cause. Tilting of the head and shoulders is often the result of astigmatism, and that the same refractive anomaly is the exciting cause of some of the cases of lateral curvature of the spine so often seen in young subjects has been shown by G. M. Gould. (See also page 558.)

Regular astigmatism is divided into five varieties, according to the relative position of the retina to the foci of the two principal meridians.

The focus of the horizontal meridian is represented by H., that of the vertical meridian by V.

1. **Simple Hyperopic Astigmatism.**—In this variety one meridian, usually the vertical, is emmetropic, and the horizontal meridian is hyperopic. The focus of the vertical meridian is on the retina; the

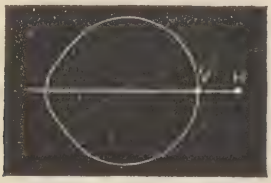


Fig. 73.—Foci of the principal meridians in simple hyperopic astigmatism.

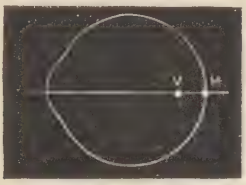


Fig. 74.—Foci of the principal meridians in simple myopic astigmatism.

focus of the horizontal meridian is behind the retina (Fig. 73); horizontal lines appear distinct.

2. **Simple Myopic Astigmatism.**—The focus of one meridian, usually the horizontal, is situated on the retina, while the focus of the vertical meridian lies in front of the retina. The vertical meridian is myopic,

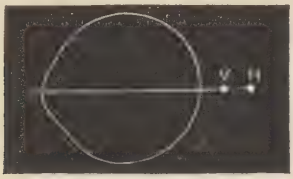


Fig. 75.—Foci of the principal meridians in compound hyperopic astigmatism.

Fig. 76.—Foci of the principal meridians in compound myopic astigmatism.

and the horizontal meridian emmetropic (Fig. 74); vertical lines appear distinct.

3. **Compound Hyperopic Astigmatism.**—All meridians are hyperopic, but usually the horizontal presents the greatest ametropia. The focus of each principal meridian is situated back of the retina, that of the vertical generally being nearest to it (Fig. 75); horizontal lines are usually most distinct.

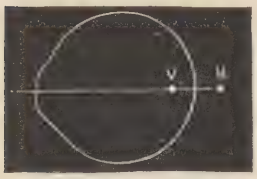


Fig. 77.—Foci of the principal meridians in mixed astigmatism.

4. **Compound Myopic Astigmatism.** — All meridians are myopic, but the vertical presents the greatest ametropia. Both principal meridians have their foci in front of the retina, that of the horizontal lying closer to the retina (Fig. 76); vertical lines are usually most distinct.

5. **Mixed Astigmatism.**—The retina lies between the foci of the two principal meridians. The horizontal meridian is hyperopic, and the vertical meridian is myopic (Fig. 77); no lines appear distinct unless the eye simulates myopic astigmatism; in this case the vertical lines appear distinct.

Recognition of Astigmatism.—Astigmatism is recognized *subjectively* by the greater distinctness of lines which run in one direction, and the blurring of those lines which run in a direction at right angles to this (see Fig. 72). The vertical strokes of a letter may appear distinct, while the horizontal strokes are hazy.

A diminished visual acuteness, unimproved by spheric lenses, in the absence of organic disease of the eye,—for example, opacity of the media or lesions of the fundus, or lesions of the visual centers,—usually is due to astigmatism. Patients frequently complain that letters have a streaked or smeared appearance; a small jet of flame seems to be drawn out in one direction.

Astigmatism is recognized *objectively* by the ophthalmoscope (see pages 108, 110), the ophthalmometer (see page 110 and Appendix), and skiascopy (see page 115).

Correction of Astigmatism.—Astigmatism may exist in a very low degree, associated with a much higher degree of hyperopia or myopia, or a marked astigmatism may exist alone, or with ametropia of the other meridians, or finally, mixed astigmatism may be present. There are several methods by which astigmatism may be measured:

1. In all cases of hyperopia or myopia, after the highest visual acuteness has been developed with spheric lenses, and even if the radiating lines on the dial appear equally distinct, a weak convex and a weak concave cylindric lens should be alternately placed in the trial-frame, in addition to the spheric lens, and their axes rotated through 180°.

If, by this maneuver, vision is improved and the patient enabled to read another line of the test-letters, astigmatism is present. For example, if the vision of a case of hyperopia of 3 D is improved by placing in front of the spheric lens a convex 0.50 D cylinder, with its axis vertical, the glass required is + 3 D sph. $\supset$ + 0.50 cyl., axis 90° or vertical; but if in the same case the maximum vision previously obtained by + 3 D sph. is not improved by the addition of a convex cylindric lens, a concave cylindric lens should be rotated through 180° in front of the spheric lens. If, under these circumstances, a concave cylinder of 0.50 D with its axis at 180° is found to improve vision and equalize the lines, the formula is + 3 D sph. $\supset$ − 0.50 D cyl., axis 180°. This result may be expressed in a simpler form by the formula + 2.50 D sph. $\supset$ + 0.50 D cyl., axis 90° (see page 33).

From this it is evident that any spherocylindric combination, in which the spheric is designated by a plus (+) and the cylinder by a minus (−) sign, unless the cylinder is stronger than the spheric, can be reduced to a simpler form, obtained by subtracting the value of the cylinder from that of the spheric lens; the difference is the strength of the rquired spheric lens. A cylinder of the same strength as the one first employed, with its sign changed to correspond to that of the spheric lens, and the axis reversed, completes the process. This method of correcting astigmatism is best adapted to those cases in which the degree is 0.75 D or less.

2. The position of the principal meridians is determined by means of the clock-face, Snellen's dial, or a series of lines, as is shown in Fig. 78.

The most distinct lines correspond to the most ametropic meridian; therefore a *stenopaic slit* is inserted in the trial-frame, in a direction at right angles to this. If vision is normal in this direction, the meridian must be emmetropic and the astigmatism is simple. The slit is then turned at right angles to its previous direction, and the glass found which

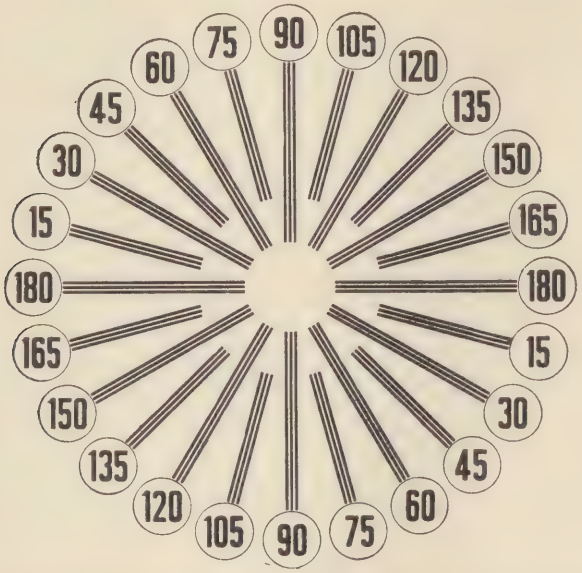


Fig. 78.—Wallace's astigmatic chart reduced to one-sixth of its diameter.

gives the highest vision. The astigmatism is represented by this glass. The following are examples:

Simple Hyperopic Astigmatism.—The patient sees horizontal lines most distinctly; the stenopaic slit is placed vertically in front of the eye: and through this $V = \frac{6}{6}$; with the stenopaic slit horizontally placed, $V = \frac{6}{9}$, with + 1 D added, $V = \frac{6}{6}$; hence + 1 D cyl., axis 90°, is the glass required.

Simple Myopic Astigmatism.—The patient sees vertical lines most distinctly; the slit is placed horizontally: $V = \frac{6}{6}$; with the slit placed vertically: $V = \frac{6}{12}$; with − 1.50 added, $V = \frac{6}{6}$; hence − 1.50 cyl., axis 180°, is the glass required.

3. The patient may not perceive any difference in the distinctness of the radiating lines until a spheric lens is placed in front of the eye, when some of them become more distinct than the others. The slit is now introduced in a direction at right angles to the distinct lines.

Vision is not normal, but a spheric lens improves it, and that lens which gives the best vision with the slit in this direction is selected. The slit is then reversed. The visual acuity is less through the slit in this position than in the previous one, and a higher lens is necessary to secure the best vision. The astigmatism is represented by the difference between the stronger and the weaker lens. This is an example of compound astigmatism, and is corrected by a spheric lens of the same strength as that which neutralizes the least ametropic meridian, and a cylindric lens equal to the difference between the two meridians. The following are examples:

Compound Hyperopic Astigmatism.—No lines appear distinct, or perhaps the horizontal ones only slightly more distinct than the others, but a convex glass makes the horizontal lines decidedly more distinct. The slit is introduced in a vertical direction: $V = \frac{6}{12}$; with + 1.50 spheric lens added, $V = \frac{6}{6}$. The slit is now turned in a horizontal direction: $V = \frac{6}{30}$; with + 3.50 D sph. added, $V = \frac{6}{6}$. The glass required for such a case is + 1.50 D sph. ⌒ + 2 D cyl., axis 90°.

Compound Myopic Astigmatism.—No lines are distinct, but a concave spheric lens possibly makes the vertical lines more distinct than the others, if the visual acuteness is not too much lowered. The slit is introduced in the horizontal direction: $V = \frac{6}{60}$; with − 5 D added, $V = \frac{6}{12}$. The slit is now placed vertically: $V = \frac{6}{60}$, − 7 D is added, and V rises to $\frac{6}{12}$. The glass required is − 5 D sph. ⌒ − 2 D cyl., axis 180°.

All that has been said in regard to the selection of glasses in myopia applies equally here. It is often impossible to correct the astigmatism accurately in the manner just described, and better results are obtained by the first method—that is, by developing the best possible vision with spheric lenses, and then adding cylinders to still further improve the visual acuteness.

Mixed Astigmatism.—Hyperopia exists in one principal meridian, and myopia in the other. Usually no set of lines appears plainer than the rest, but the addition of a concave or convex spheric lens brings out some lines more distinctly than the others. Thus a clue to the principal meridians is obtained. With the slit before the eye, a convex spheric lens is placed in position and the slit rotated until the vision becomes more distinct. The hyperopic meridian has then been found.

Example.—Suppose the hyperopic meridian to be horizontal and V to be most improved by + 3 D. The slit is turned to the vertical position, and it is found that a − 4 D gives the best vision. The difference between these two meridians is 7 D. A + 7 cylinder, axis 90°, placed before such an eye would produce a myopia of 4 D, while a − 7 cylinder, axis 180°, would produce a hyperopia of 3 D, consequently with the + 7 cylinder we must associate a − 4 spheric lens, and with the − 7 D cylinder a + 3 D spheric lens. Such a case could be corrected by either of the following formulas: + 3 D sph. ⌒ − 7 D cyl., axis 180°; or − 4 D sph. ⌒ + 7 D cyl., axis 90°; or by means of two cylindric lenses with their axes at right angles to each other, viz., + 3 D cyl., axis 90° ⌒ − 4 D cyl., axis 180°.

Dr. J. S. Johnson,[1] of St. Paul, employs a method of determining astigmatism which he calls "the reversal of the clock-dial chart" when it is viewed through successive spheric lenses. The first indication of such a change marks the dividing line between the hyperopia and the astigmatism and between the spheric and cylindric correction. If carried carefully to the point of complete reversal, it will also show the ametropia of highest degree and thus serve all the purposes of the stenopaic slit.

Thorington also utilizes this method in determining the acuteness of vision, which he maintains under definite conditions is an index of the strength of the spheric lens which will give normal vision. For this purpose he has designed a series of "metric test-letters and lines."

The *astigmatic lens*, or *crossed cylinder*, as pointed out by Edward Jackson, is most useful to determine the amount and also the principal meridians of astigmatism. This astigmatic lens is employed as a supplementary lens; the axis of the cylinder is first placed in the same direction as the axis of the cylinder in the trial-frame, and next it is turned perpendicular to it. In one position it enhances the effect of the cylinder in the trial-frame; in the other it diminishes the effect. Therefore, if the vision is unchanged by an astigmatic lens in either of these positions, the cylinder in the trial-frame is correct. If the vision is improved by the astigmatic lens when placed in one position, but not made better in the other position, the cylinder in the trial-frame must be changed accordingly.[2] Various astigmatic lenses may be used having a value of 1 D (+ 50 cyl. $\supset$ − 0.50 cyl.), 0.50 D, and 0.25 D.

The following additional facts concerning lenses require mention: If a spherocylinder is in position before an eye, and vision is improved by placing before it another cylinder of the same sign (+ or −), with its axis at right angles to that of the first, it shows that a stronger spheric and weaker cylinder are required.

If vision is improved by placing in position another cylinder of the same sign, with its axis parallel to the first, it shows that the same spheric with a stronger cylinder should be adopted.

If vision is improved by placing in position another cylinder of different sign, with its axis parallel to the first, it shows that a weaker cylinder with the same spheric lens is needed.

If vision is improved by placing in position a cylinder of different sign, with its axis at right angles to the first, it shows that a weaker spheric lens with a stronger cylinder must be employed.

4. Astigmatism is best estimated and the correcting glass determined by *objective methods:* skiascopy and the ophthalmometer. These have been referred to and are elsewhere explained (see pages 110 and 111). All methods should be tried before the glass is finally ordered, and the highest visual acuteness possible should be obtained.

Ordering of Glasses.—Glasses are ordered for astigmatic eyes according to the general rules already given. For distance, the full

[1] Ophthalmic Record, October, 1901.

[2] For full directions in regard to the use of the lens see article by E. Jackson, Ophthalmic Record, August, 1907.

correction is ordered in myopic astigmatism and usually in mixed astigmatism; in compound hyperopic astigmatism the spheric lens is usually weakened to meet the requirements of accommodation, but the full cylindric lens should be ordered. In simple hyperopic astigmatism it may be necessary to add a concave spheric lens; thus, if the correction under full mydriasis at 4 meters should prove to be + 1.50 D cyl., axis 90°, the formula for the glass to be worn after return of accommodation would be − 0.25 D sph. ⌒ + 1.50 D cyl., axis 90°. In compound myopic astigmatism the spheric lens is sometimes weakened for near work. Simple myopic astigmatism and mixed astigmatism give an opportunity for simplifying reading-glasses, as will be described under Presbyopia.

At present there is no uniform plan for the designation of the meridians in astigmatism, and, consequently, formulas for glasses intended to correct astigmatism do not have a uniform meaning in all parts of the world. Drs. Thomson and Harlan[1] have conveniently summarized three systems as follows:

1. The zero is placed at the end of the horizontal meridian to the patient's left, and the degrees are counted on the upper semicircle to 180° at his right.

2. Zero is placed at the top of the vertical meridian, and the degrees are counted to the nasal and temporal sides to 90° at the horizontal meridian.

3. The zero mark is placed at the nasal extremity of the horizontal meridian in each eye, and the degrees are counted on the upper semicircle to 180° at the temporal extremity.

The first is the one in almost universal use in this country, the formula for the glasses being written in accordance with the markings on the trial-frame.

Irregular Astigmatism.—A low degree of this defect exists in nearly all eyes, but it does not interfere with good vision. When its degree is increased by irregularities of the corneal surface from ulcers and cicatrices, the vision is very much reduced, and where such lesions are extensive, optical therapeutics may be unavailing. Often, however, within the pupil space small areas may be found in which the refraction is tolerably uniform, and vision may be decidedly improved by lenses—spheric and cylindric. All such eyes should be carefully studied by objective methods, and full trial with lenses should be made. Stenopaic spectacles render vision more distinct, but they embarrass the wearer by limiting the field of vision. An iridectomy sometimes improves vision by displacing the pupil toward a more regular portion of the cornea.

Surgical Treatment of Astigmatism.—It has been proposed to correct astigmatism by incising the cornea with a Graefe knife, or by producing a wound two-thirds of the depth of the cornea with the galvanocautery (Laus). The operation should be performed on the

[1] Archives of Ophthalmology, 1893, vol. xxii, pp. 251–261. This paper contains an excellent discussion of this subject and an analysis of the arguments for the various systems.

meridian of greatest refraction (Borsch). The author has had no experience with these procedures.

Anisometropia.[1]—This term includes cases in which one eye is much more hyperopic or myopic than its fellow, or where one eye is astigmatic and the other not, or where myopia exists in one eye and hyperopia in the other. No general rule for the management of cases of this character can be given, but the author agrees with Duane that "in the majority of cases of anisometropia, even those in which the difference in refraction exceeds 2 D, the full correction can be applied with success." The patient, however, must be required to wear the glasses constantly, and must be willing to bear with temporary discomfort while the eyes are becoming accustomed to the lenses. The causes which give rise to discomfort may be summarized as follows: Diplopia and asthenopia from the unequal prismatic effect of the unequally strong lenses; diplopia from imbalance of the ocular muscles, with the full correcting lenses the double images being more manifest; and difficult binocular vision because the retinal images of the two eyes are of a different size, a cause, however, which is considered fallacious by Duane. Exophoria and hyperphoria are often associated with anisometropia; squint may be caused by this refractive condition and may be materially improved by the use of the correcting lenses. If discomfort ensues, success may follow the attempt to train the function of the more defective eye by temporarily excluding the other from vision.

Presbyopia.—The accommodation diminishes gradually from early life onward, and the near-point recedes farther from the eye with each succeeding year. When by this recession the near-point reaches a distance of 30 to 40 cm. from normal eyes, it interferes with their use at close range, and convex lenses are usually required. *Presbyopia* has now begun, and is a normal result of growing old.

Causes.—The cause of presbyopia consists in loss of the elasticity of the crystalline lens, which is thus prevented from assuming the increased convexity which constitutes the essential factor of accommodation. This increase of convexity, necessary for seeing near objects, must be supplied to the eye by a suitable lens.

Presbyopia usually begins in emmetropic eyes at the age of forty-five. Unusual visual acuteness, or vigor of accommodation, however, may enable a person to dispense with glasses for several years longer. A visual acuteness of $\frac{6}{4}$ permits its possessor to see the same object distinctly at 30 cm., which another individual with a vision of only $\frac{6}{6}$ would have to hold at 20 cm. Patients occasionally postpone the time of wearing reading-glasses by holding fine print in a bright light, the resulting contraction of the pupil rendering vision more distinct. Presbyopia is to be distinguished from hyperopia, which is often latent and

[1] This term, according to Suker, is often inaccurately applied. He would employ it only to describe an unequal amount or degree of the same kind of refractive error in the two eyes. To describe a different kind of refraction in the two eyes he prefers the word *antimetropia*.

confounded with it. Correction of hyperopia restores the far-point of the eye to infinity.

Correction of Presbyopia.—In the first stages of presbyopia, while considerable accommodation still remains, a weak convex lens is required, which enables the person to see near objects by rendering the rays less divergent, as if they came from a somewhat greater distance.

There is still a range of vision from the focal distance of the glass to the near-point. A person who has an accommodation of 3 D, and requires + 1.50 D in addition, will have a range from the focal distance of the glass $\frac{1 \text{ meter}}{1.50} = 66$ cm. to his near-point through the glass; 3 D + 1.50 D = 4.50 D; $\frac{1 \text{ meter}}{4.50} = 22$ cm.

When the accommodation is entirely obliterated at seventy-five years of age, the convex glass must be stronger. The rays are now rendered parallel, as if they came from an infinite distance, and the object must be held at the focus of the lens. There is, therefore, no range of vision.

The presbyopic glass is estimated after the eye has been rendered emmetropic by neutralizing any hyperopia or astigmatism which may be present (for the management of myopia and myopic astigmatism in these circumstances see page 151).

The *near-point* of vision should be carefully determined for each eye separately. The ability to read 1-meter type at 30 cm. is not equivalent to the act of accommodating for 30 cm.; in order fairly to accommodate for 30 cm. the patient should be able to read type which represents normal vision at 30 cm. (see page 38). If the accommodation is normal, the near-point will correspond closely with the figures given in the table. The additional refractive power required may then be calculated. Unduly strong glasses should not be employed in approximating the near-point, lest the far-point be brought too close and serious discomfort ensue. Most persons read at an average distance of from 33 to 40 cm., and in early presbyopia considerable range of vision exists on either side of these points; but at sixty years and later there is little play —the near-point and far-point are close together. A glass with which the patient reads easily at 33 to 40 cm. may then be ordered, unless visual acuteness is much diminished.

Table of the Position of Near-point at Different Ages

Age.	Accommodation.	Point.
45	3.50 diopters	29 cm.
50	2.50 "	40 "
55	1.75 "	57 "
60	1 "	100 "
65	0.50 "	200 "
70	0.25 "	400 "
75	00 "	∞

At the age of forty-five it is usually necessary to supply a + 1 D spheric lens for reading, provided the eye is emmetropic; if the eye is

hyperopic, 1 D + the correction for the hyperopia; if myopia exists, + 1 D is not required. Plus 1 D added to the 3.50 D of accommodation which the eye possesses at forty-five years = 4.50 D; this brings p to 22 cm. $\left(\frac{100}{4.50} = 22\right)$, and r to 100 cm.

At fifty years of age + 2 D is usually required, with the same modifications in case of hyperopia or myopia. This glass, added to the accommodation which the eye possesses at 50,—viz., 2.50 D,—also makes 4.50 D; this brings p to 22 cm., but r is now only 50 cm. distant.

At fifty-five years, + 2.50 D is the glass usually required, which, added to the accommodation (1.75), gives a refractive power of 4.25 D; p = 23.5 cm., r = 40 cm. If stronger lenses than this are used, r is brought still closer, and the patient is forced to hold the book near his face. If $V = \frac{6}{6}$, it is not necessary to order a glass stronger than the one recorded; indeed, usually + 2.00 D or 2.25 D is sufficient, as most persons prefer a glass which enables them to read, resting the book on the lap or the arm of a chair. These glasses are for emmetropic eyes. In hyperopia with presbyopia they are to be added to the hyperopic correction. Visual discomfort (asthenopia) often develops with the first attempts to use presbyopic glasses, owing to the development of temporary convergence—insufficiency from relaxation of accommodation. In these circumstances it is usually necessary to reduce the strength of the glass.

As visual acuteness diminishes, a stronger lens is necessary to enable the object to be held closer, and thus subtend a larger visual angle. The glass may be increased to 4, 5, 6, or even D 8. The strong glasses necessitate the close approximation of the object and a corresponding diminution in the field of vision. The only rule in the selection of such glasses is to give that glass which affords the necessary vision with the least inconvenience. With very great diminution of sight, requiring glasses of 8 or 10 D, binocular vision is impossible, and the better eye should be supplied with a correcting glass, and the other excluded from vision.

With binocular vision, the reading-glasses for the two eyes should be equal in strength; consequently, when a different degree of ametropia exists in the two eyes, a corresponding difference should be made in the reading-glasses. Occasionally, in the absence of ametropia, or even after its correction, when present, there is an inequality of the accommodative power in the two eyes. Thus, a patient of fifty years may have 2.50 D of accommodation in the right eye, and only 1.50 D of accommodation in the left. Under such conditions it is usually necessary to order a correspondingly stronger reading-glass for the eye with the weaker accommodation.

Frequently, modifications are required in the strength of the glass to suit particular vocations—for example, reading music, reading in the pulpit, working at a bench, playing cards, etc. In these circumstances it is necessary to ascertain the distance from the eye at which the work

is placed, and to order a glass whose focal distance is not less, but, if possible, somewhat greater than the distance required. Not infrequently the patient must be provided with two sets of glasses—one pair for the ordinary reading distance and another of greater focal length for work at a longer range. Thus, a patient of fifty-five years may require + 2.25 D for reading, but for playing the piano + 1.25 D. This correction of accommodative defects at what may be called an intermediate distance is most important, and the character of the patient's work must always be carefully ascertained before ordering a glass.

In myopia, myopic astigmatism, and mixed astigmatism the rules for the selection of reading-glasses require particular mention. Patients with low degrees of myopia, not higher than 2 D, do not need reading-glasses at as early an age as emmetropic or hyperopic subjects. The amount of myopia may be considered the equivalent of the convex glass suitable for the correction of the presbyopia. A myopia of 1 D, consequently, would enable a person to attain the age of fifty without the necessity of reading-glasses. At that age he would require + 1 D for reading, and at fifty-five + 1.50 D, and at sixty, possibly + 2 D, depending upon his visual acuteness. A myope of 2 D could dispense with reading-glasses until the age of fifty-five (often until a later period); then he would require + 0.50 D; at sixty, possibly + 1 D. A myope of 3 or 4 D never becomes presbyopic in the ordinary sense; he can read at any age without glasses.

In higher degrees of myopia it is necessary to order a concave glass from 2 to 5 D less than the full correction. The age has little influence on the amount of reduction; myopes readily relax accommodation; the degree of myopia and the visual acuteness are the two important factors. A concave glass is given which will extend the far-point to a comfortable distance. A myope of 6 D would probably require from − 3 to − 4 D for reading; a myope of 10 D, about − D, and a myope of 15 or 20 D would require a reduction of 5 or 6 D from the full correction. In these high grades vision is much reduced, print cannot be seen unless held close to the eye, so that extension of the reading distance is out of the question. The farthest point at which a book can be read should be determined, and a glass given of the same length of focus. Prisms are often necessary. When the vision is much reduced, myopes usually read with one eye without the aid of any glass.

A patient with simple myopic astigmatism generally should be provided with a convex cylinder of the same number, its axis being reversed. Thus, a patient whose myopic astigmatism is corrected by − 2 D cyl., axis 180°, will be comfortable with a + 2 D cyl., axis 90°. This glass produces a myopia of 2 D in all meridians, and because the patient has been accustomed to see through a myopic meridian, he prefers this glass to the concave cylinder, which makes him accommodate. As a rule, simple myopic astigmatism may be utilized to determine the reading-glass in patients who have reached the age of thirty-five, provided its degree is not too high. A convex cylinder of a strength equal to the concave cylinder with its axis reversed will be sufficient.

If the degree of myopia thus produced is too great for comfortable reading, a concave spheric lens may be added to the convex cylinder. Thus, an astigmatic eye corrected by a − 4 D cyl., axis 180°, would probably require − 1.50 D sph. ⌒ + 4 D cyl., axis 90°.

If the degree of astigmatism is unequal in the two eyes, a spheric lens is required over one eye to equalize the refraction. For example:

1. R. E. − 5 D cyl., axis 180°. L. E. − 3 D cyl., axis 180°. This case requires a − 2 spheric lens to be added to the right eye—viz., − 2 D sph. ⌒ + 5 D cyl., axis 90°, to make its refractive power equal to that of the left, + 3 D cyl., axis 90°.

2. R. E. − 1 D cyl., axis 180°. L. E. − 2.50 D cyl., axis 180°. In this instance, according to the circumstances, age, etc., one of the following combinations may be ordered: R. E. + 1 D cyl., axis 90°; L. E. − 1.50 D sph. ⌒ + 2.50 D cyl., axis 90°; or R. E. + 1.50 D sph. ⌒ + 1 D cyl., axis 90°; L. E. + 2.50 D cyl., axis 90°. Both of these combinations equalize the refraction of the two eyes, the first by producing in each eye a myopia of 1 D, the second a myopia of 2.50 D.

In cases of compound myopic astigmatism, should the myopia amount to several diopters, the reading-glass is secured by a sufficient reduction of the strength of the spheric without change of the cylindric lens.

If, in lower degrees of compound myopic astigmatism, it is desirable to increase the refraction one or more diopters, the procedure is somewhat different. Thus, if the combination is − 0.50 D sph. ⌒ − 1 D cyl., axis 180°, and the spheric lens is omitted, + 0.50 D is gained; by substituting for the concave cylinder a convex cylinder with its axis reversed, an additional gain of 1 D is secured; + 1 D cyl., axis 90°, in this case is equivalent to adding + 1.50 D sph. to the original combination. If still more refractive power is desirable—*e. g.*, + 2 D, + 0.50 D sph. ⌒ + 1 D cyl., axis 90°, gives the additional amount.

In another combination, − 0.75 D sph. ⌒ − 4 D cyl., axis 180°, it is desired to add + 2.50 D for reading. Dropping the − 0.75 D spheric lens, + 0.75 D of refractive power is obtained; substituting for the concave cylinder, convex 4 D cyl., axis 90°, + 4 D more are gained, making + 4.75 D. This is too high, hence it would be necessary to combine − 2.25 D sph. ⌒ + 4 D cyl., axis 90°, in order to obtain the desired + 2.50 D. A simpler method of procedure in this case would be to drop the − 0.75 D spheric lens; the uncorrected myopia would then furnish 0.75 D of the requisite 2.50 D, leaving 1.75 to be obtained. A + 1.75 D added to the − 4 D cyl., axis 180°, would make the proper combination.

In mixed astigmatism, a combination of spheric lens and cylinder is usually employed, and by using a concave spheric and convex cylinder the combination necessary to produce any additional refractive power can easily be found.

If the myopia produced by the convex cylinder alone is greater than the power of the lens it is desired to add, a concave spheric lens equal to the difference is given, thus: To the combination − 3 D sph. ⌒ + 5 D

cyl., axis 90°, it is desirable to add + 2 D. − 3 + 2 = − 1, hence − 1 D sph. ⌒ + 5 D cyl., axis 90°, is the glass required. Again, to − 1 D sph. ⌒ + 3 D cyl., axis 90°, it is desirable to add + 2.50 D. − 1 + 2.50 = + 1.50, hence + 1.50 D sph. ⌒ + 3 D cyl., axis 90°, is the necessary glass. The myopia is in this case insufficient.

It is a point of some importance, in ordering reading-glasses containing cylindric lenses, to give attention to the relation of the axes of the cylindric lenses. It has been assumed, for the sake of simplicity, that the axes of convex cylinders are placed at 90° and the axes of concave cylinders at 180°; this is commonly so, but the exceptions are numerous. It is a frequent condition in astigmatism to have one principal meridian inclined 15° to the right of the vertical in one eye, while the meridian of the same refraction in the other eye is inclined the same amount to the left of the vertical. This produces no serious disturbance in wearing the glasses if they are properly centered, although at first a rectangular figure appears like a rhombus. In a little time the eyes adapt themselves to the glasses, and this appearance is lost.

When the meridians of similar refraction are at greater angles than this, especially if the cylindric lenses are strong, there is often inconvenience in wearing them on account of the prismatic deviation and the unequal distortion of objects which cylindric lenses produce. Occasionally the axes are as much as 90° apart, one at 45° and the other at 135°, or one at 90° and the other at 180°. The glasses now deviate rays from an object in different directions, according as the eye looks through the glasses above or below the optical centers, or to the right or left of them. Such a case would be represented by + 3 D cyl., axis 180°, in right eye, and + 3 D cyl., axis 90°, in left eye. The difficulty is not obviated by ordering a formula like the following: R. + 3 D cyl., axis 180°, L. + 3 D sph. ⌒ − 3 D cyl., axis 180°, because the same displacement results. It will be found that the best solution of this difficulty is to ascertain the distance from the eye at which the person usually holds the book, and the relative position it occupies to the eye. The direction of the visual lines may thus be determined, and the optical centers of the glasses should be so placed that the visual lines will pass through them. There is then no deviation. Of course, this renders necessary a separate pair of glasses for reading. When cylindric lenses with axes in unusual directions are required for distance, the optical centers should bear the same relation to the visual lines in distant fixation. These disturbances are aggravated by removing the glass farther from the eye, and, conversely, the trouble diminishes as the glass is brought nearer to the eye.[1]

Bifocal Lenses.—When presbyopic patients require two sets of glasses, one for distance and one for reading and close work, it is the custom, instead of providing them with separate sets of glasses, prescribed according to the rules set forth in the preceding paragraphs, to

[1] Consult interesting papers in the Archives of Ophthalmology, vol. xviii, by Dr. J. A. Lippincott; in the Ophthalmic Record, vol. i, No. 1, by Dr. G. C. Savage; and Dr. R. J. Phillips, in the Annals of Ophthalmology, vol. ii, p. 31.

order *bifocal lenses* (Fig. 79). By means of such glasses the inconvenience of changing spectacles is avoided, and, moreover, the patient's eyes are constantly adapted by proper lenses to close and long ranges. A spheric lens, suited to needs of accommodation, ground very thin, is

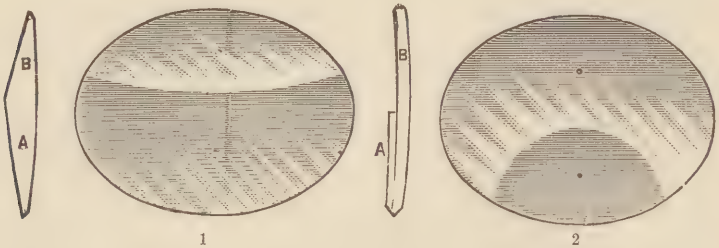


Fig. 79.—Bifocal lenses: 1, Solid bifocal lenses; 2, cemented bifocal lenses.

cemented on the lower portion of the distance glass, usually upon its inner side. The size of the additional segment varies. Generally one $12\frac{1}{2}$ mm. in height and 22 mm. in length is sufficient. The shape of the supplementary lens varies. Commonly it is an oval; sometimes it is made in the form of a circle, and sometimes it is dome shaped. Solid and single crystal bifocal lenses are also manufactured. A particularly graceful form of bifocal lens has been designed by J. L. Borsch, of Philadelphia (Fig. 80). In this, for the usual presbyopic segment, there is substituted a small lens 15 mm. in diameter, made of flint glass and sunk into the distance lens, which is made of crown glass. The increased refraction of the small lens depends upon the higher index of the flint glass. Its exposed surface is ground to the same curvature as that of the larger lens. Lenses of this character are usually known as "concealed" or "kryptok" bifocals.

Fig. 80.—Borsch's bifocal lenses.

SPECTACLES AND THEIR ADJUSTMENT

After the refraction of the eye has been determined and the proper combination of lenses selected, the glasses should be properly ground, mounted in spectacle-frames, and correctly adjusted to the patient's eyes. In place of spectacle-frames so called "eye-glasses" are much employed; if they can be firmly adjusted, and the spherocylindric combination is not of high degree, there is no serious objection to their use, although they can never be as accurately applied as spectacles. Patients should not be allowed to wear glasses until the surgeon has satisfied himself that the formula for the lenses has been faithfully followed by the manufacturing optician.

In order to do this he proceeds as follows: If a simple spheric lens has been ordered, this and a spheric lens from the trial-case, of the same number but opposite refractive character, are placed in close contact and some distant object observed through the combination, while the glasses at the same time are gently moved up and down and to and fro. If the glass is correct, this maneuver has no influence upon the size or position of the object, which appears exactly as it would if it had been looked at through a piece of plane glass. The glasses are then said to neutralize each other. If the lens ordered does not neutralize the test-glass from the trial-box, a weaker or stronger number is tried until the glass is found which produces complete or nearly complete neutralization. Thick bispheric lenses of different refractive character will not neutralize each other entirely, even if they are of the same number. The convex lens always preponderates. With a suitable "lens-measure" the character of a lens can be quickly determined.

If a cylindric lens has been ordered and has been correctly ground, it will be neutralized by a cylinder of the same number but of opposite refraction, with its axis turned to the same angle as that of the lens ordered. On shaking these two lenses, which are placed in contact, there should be no motion of the object viewed through them. The direction of the axis of a cylinder may be determined by finding the position in which the lens may be shaken without producing any motion of the object. For example, if the axis of the cylinder is vertical, no motion in the object looked at would occur when the spectacle lens is moved up and down. A line drawn on the glass with a pen marks this, and by placing the lens thus marked on a *protractor*, the degree of the angle may be read off.

A combination of spheric and cylindric lenses is to be tested by a spheric lens held on the spheric surface of the spectacle lens, and a cylindric lens held on the cylindric surface of the spectacle lens, and proceeding in the manner just described.

The *optical center* is ascertained as follows: The lens is held by its edges between the finger and thumb, and, care being taken not to hold it obliquely, it is passed from right to left until the test-object (a vertical line) forms a continuous line above the lens, through the lens, and below the lens. If the axis of the lens is not exactly in line with the test-object, the part seen above and below the lens will not coincide with the part seen through the lens. When a continuous line is obtained through the lens with the object above and below, the lens should be marked with a line drawn across its surface over the part where the line is seen, just as the outline of a figure is traced on a transparent plate. The glass is now turned around so that the line is at right angles to its former position; another portion of the lens is found through which the test-object is also seen in a continuous line with the part above and below. This is traced on the glass with ink, and the intersection of the two lines thus traced marks one extremity of the axis of the lens. In most lenses the distance from the surface to the center is so slight that we may consider this point on the surface as the center, and each

lens should have its center marked by a dot of ink. Strong lenses may be centered more easily by using the window-bars, while the glass is held close to them, or the edge of a card or sheet of paper, which is laid on the desk. Still greater accuracy may be obtained by using a card on which two lines are drawn, crossing each other at right angles; both principal meridians may in this way be found at once; the optical center then lies over the intersection of the lines.

The spectacles should now be placed on the patient, and the position of these centers in relation to the pupil carefully noted. The patient is first asked to look across the room; the centers of the pupils should correspond with the dots on the glasses. Next, the patient is required to look at the finger of the surgeon held at 40 cm. distance, and it will be noticed that the centers of the pupils and the dots no longer coincide, but that the former have passed to the inner side of the latter. If the glasses are for distance or for constant wear, the space between the centers of the lenses should be the same as the interpupillary distance; if the glasses are for reading alone, the distance between the centers must be lessened. The ordinary reading distance being 40 cm., the visual lines converge to this point, and the farther the glasses are from the center of rotation, the nearer the centers should come to each other; therefore it is necessary to make the distance between the centers of the reading-glasses from 2 to 4 mm. less as compared with those of distance glasses, so that the visual lines may pass through these centers. Thus: The center of the pupil deviates inward about 1 mm. in fixing at a point 40 cm. distant, as the pupil is 11 mm. in front of the center of rotation; a glass placed 13 mm. in front of this would require its optical center to be 1 mm. farther inward than the pupil—2 mm. in all. The two centers should thus be 4 mm. nearer together in reading-glasses than in those for distance.

Should glasses be ground with badly placed centers,—that is, too far apart or too close together,—the most unpleasant consequences may arise: obstinate diplopia, severe neuralgia, headache, and tendency to squint.

The patient should observe some distant object while the interpupillary distance is measured during distant fixation, fixing his eyes on the finger-tip of the observer, held about 30 cm. from his eyes while the measurement is noted during convergence. There should be a variation of 2 mm. between these two measurements. If the difference is greater than this, there is a probability that the patient has an insufficiency of convergence, and, in this case, the centers of convex glasses should be brought closer together; those of concave glasses placed farther apart. In order to ascertain the amount of deviation which is produced by decentering a spheric lens, see page 20.

Reading-glasses should be tilted forward and placed about 5 mm. lower than those for distance, in order to conform with the depression of the visual line in reading. Spectacles are always to be preferred; but the prejudice of many patients in regard to spectacles will often have to be respected. The tilting forward of eye-glasses is rather an advantage

in reading, and in myopia the effect of this tilting is equivalent to a cylindric lens with a horizontal axis. This fact accounts for the preference shown by some patients for a simple concave spheric uncombined with a cylindric lens, in spite of the existence of a slight degree of astigmatism.

When separate glasses are required for distance and reading, it is often very inconvenient to make the change from one to the other. The two glasses may be combined in the same frame by making the lower half suitable for reading and the upper half for distant vision (Franklin or split bifocals). *Bifocal lenses*, as already described, constitute a more suitable arrangement (see page 153). "Hook fronts" are very convenient for making a rapid change from reading to distant vision, or "half-hook fronts" may be employed. Occasionally, for special purposes, *trifocal lenses* are manufactured—that is, an upper segment correcting the distant vision and a lower segment correcting the close vision are cemented in a lens which corrects the intermediate vision.

CHAPTER V

DISEASES OF THE EYELIDS

Congenital Anomalies.—Complete absence of the lids (*ablepharia totalis*), or their partial development (*ablepharia partialis*), is a rare anomaly. If the defect is of such a nature that the lids are wanting and the orbit divested of any covering for the globe, the condition is designated *lagophthalmos*, a name which also, and perhaps more properly, has been given to a contracted state of the eyelids preventing their closure, independent of any muscular paralysis.

Cryptophthalmos is a condition in which neither eyelid nor conjunctival sac is present, but the exterior integument passes in front of, and buries an eye more or less developed.

Fig. 81.—Coloboma palpebræ and anophthalmos.

Cleft eyelid (*coloboma palpebræ*) is a fissure, in appearance not unlike a harelip, which may be confined to the upper lid (its most common situation), but which also has been noted in the lower lid (when the upper lid of the same eye is always also involved), and even in the upper and lower lids on each side. The center of the cleft may contain an intervening membranous portion, either movable or pressed against the cornea (Fig. 81), or may be clear, so that the cornea fits exactly into it when the eyes are directed straight forward (Posey).

Coloboma of the eyelids is most frequently associated with harelip and cleft palate; rarely with facial defects and other congenital anomalies in the eyeball. The deficiency may be remedied by a plastic operation.

Symblepharon, or a cohesion, either partial or complete, between the eyelid and the ball, and *ankyloblepharon*, or a union between the margins of the lids, are unusual congenital anomalies. Sometimes only the middle portions of the lid-borders are attached by a filamentous band, or the outer angles of the lids adhere, and produce the defect known as *blepharophimosis*.

Ectropion, or eversion of the edges of the eyelids, is a rare condition usually accompanied by increased size of the eyeball. *Entropion*, or inversion of the edges of the eyes, which in slight degree is said to be normal before birth, has been found associated with *distichiasis*, or the development of supplementary incurved eyelashes.

In congenital distichiasis, Kuhnt has demonstrated that the second row of lashes consists of fine hairs springing from the posterior part of the intermarginal area. Meibomian glands are wanting, and their

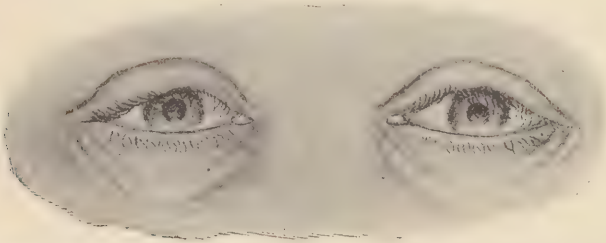


Fig. 82.—Congenital distichiasis (from a patient in the Philadelphia General Hospital).

places are taken by the abnormal cilia. Occasionally the condition appears to be hereditary.

The operations which are employed to rectify these conditions when of pathologic origin (see page 605) are also applicable here.

Epicanthus is a striking congenital anomaly giving rise to an apparent convergent strabismus, owing to the passage of a fold of skin from the inner end of the brow to the side of the nose, covering the internal canthus, its free concave border stretching outward. Thus the caruncle, lacrimal punctum, and, in aggravated forms, a considerable portion of the area of the lids, are hidden. Epicanthus generally is bilateral and is usually associated with ptosis (Fig. 83). The same condition in minor degrees is often seen in newborn children, and disappears with the subsequent development of the face and nose. A similar fold of skin at the outer commissure of the lids has been described, the so-called *external epicanthus*. True epicanthus may be hereditary, and the defect has been noted in several generations (von Hippel).

Fig. 83.—Epicanthus and congenital ptosis (from a patient in the Children's Hospital).

Epicanthus may be remedied by excising a portion of the redundant integument from the bridge of the nose, and stitching together the opposed surfaces.

Epitarsus is a somewhat wing-shaped duplicature of conjunctiva which passes from the fornix to be inserted near the lid border, and is so undermined that a small probe can be passed beneath it (Schrapringer). This anomaly is also described under the name *congenital pterygium.*

Congenital ptosis consists in a drooping of the upper lid over the eyeball. It may be single or double, but never amounts to complete closure. In one variety there is an actual redundancy of the lid tissue; in the other the lid is thin and the skin stretched, owing to imperfect development or absence of the levator palpebræ. It may be caused by a nuclear and, rarely, by a cortical lesion.

This anomaly is often associated with other vices of conformation, especially epicanthus, and with paralysis of the exterior ocular muscles, especially the superior rectus. It may be corrected by one of the operations described on page 600.

Congenital *fistula* of the upper lid has been reported. A *supernumerary eyelid*, presenting as a small growth at the inner canthus, has been described (J. L. Shoemaker and A. Alt).

Edema of the lids in general terms is *inflammatory* or *non-inflammatory* in origin. It may result from traumatism, the sting of insects, and is seen with severe inflammations of the conjunctiva, of the cornea, the uveal tract, and especially with infections of the globe, *e. g.*, panophthalmitis and in association with acute glaucoma. It is a common symptom of general conditions (renal or cardiac), and is conspicuous in thrombosis of the cavernous sinus, disease of the sinuses, especially of the ethmoid and antrum, and sometimes occurs in a fugitive, and not infrequently recurrent, form. The last variety has ben observed with migraine, at the time of the establishment of menstruation, and spontaneously without apparent cause. Some cases are analogous to urticaria. The eyelid is a common seat of *angioneurotic edema*. Some types of edema, non-traumatic in origin, have been called *essential edemas*. According to Trousseau, they are often *arthritic* in origin.

A condition has been described characterized by great symmetric swelling of both eyelids which present the usual appearances of chronic edema, and to which Sir Anderson Critchett has given the name *solid edema of the eyelids*. The affection has been regarded as a recurrent lymphangitis of lupoid origin (Morris). Not infrequently the patients have a history of repeated attacks of erysipelas (Eyre). Persistent, non-inflammatory edema of the lids is sometimes observed in children following measles (Lawson and Sutherland). In rare instances lid edema is a late symptom of syphilis.

Erythema of the lids appears in the form of a hyperemia, more or less diffused, under the influence of heat (sunburn), traumatism, and irritating poisons, or as symptomatic of a systemic disturbance.

A *passive hyperemia*, in which the superficial veins of the lids are dilated and the tissue red and slightly swollen, commonly is the result of prolonged bandaging of the eye, and is seen in an *active* state associated with most of the inflammatory diseases of the cornea and conjunctiva.

Urticaria, or **hives,** appears in the form of characteristic wheals associated with much tingling and burning sensation.

Treatment.—This consists in removal of the cause and the application of a soothing lotion—lead-water or extract of hamamelis.

Erysipelas rarely attacks the eyelids as a primary affection, but spreads to them from the contiguous facial area. The chief danger of the affection in this region is its liability to infect the tissues of the orbit, producing compression of the central vessels of the retina and blindness. It may spread to the membranes of the brain and be fatal. The characteristic red, shining, and later brawny swelling, and the formation of cutaneous vesicles and small abscesses, are the symptoms which establish a diagnosis.

Abscess of the lid (**phlegmon**) appears as a localized red elevation, and is often a severe form of furuncle or hordeolum. The entire lid is hyperemic and the conjunctiva injected and often edematous. There are much pain, headache, and fever. This affection is provoked by injury, exposure, and disease of the orbit, and sometimes arises without ascertainable cause, especially in debilitated people and children. In rare instances it has been followed by thrombosis of the orbital veins and cavernous sinus and has terminated fatally.

Treatment.—Pointing should be favored by hot, slightly carbolized fomentations or compresses soaked in boric acid solution. As soon as fluctuation is detected, or even earlier, a sharp knife may be thrust through the swelling, parallel to the muscle-fibers, and the contents evacuated; the cavity is to be kept clean with an antiseptic fluid.

Furuncle of the lid is a localized inflammation of the skin and subcutaneous tissue, presenting symptoms analogous to abscess, which goes on to the formation of a central slough or "core." The surrounding and overlying tissue may become gangrenous in subjects of poor nutrition.

Malignant pustule, or *specific anthrax*, caused by the entrance of the *Bacillus anthracis*, and *malignant edema*, or a form of *spreading gangrene*, occasionally attack the eyelids. The former usually arises among persons whose occupation brings them in contact with diseased animals or decayed animal matter; the latter may follow an injury, influenza, the exanthemata, typhoid fever, whooping-cough, and erysipelas, but has also been described as an idiopathic affection. Sometimes gangrene of the lids is metastatic in origin and occurs during pyemia. *Noma of the eyelids*—that is, a symmetric gangrene of the lids and region of the lacrimal sac—has been described.

Treatment.—According to the condition present, this should include incision, promotion of the separation of the sloughs by hot compresses steeped in boric acid solution, the use of the actual cautery to check the destructive tendency, and antiseptic lotions.

Usually staphylococci and streptococci are present, but in some of these gangrenous processes diphtheritic bacilli have been found, and under such conditions serum therapy would be strongly indicated.

Blastomycosis of the Eyelids.—Blastomycotic dermatitis, which may affect any portion of the body, has involved the eyelids in about one-fourth of the cases thus far reported (Casey Wood). The affection begins as a red papule and gradually extends until it forms a flat, wart-like growth with a red elevated margin. Dry crusts are apt to cover

its surface, and on their removal a bleeding rough surface is evident. Miliary abscesses are seen in the softened edges, from which and from the surface of the growth a mucopurulent discharge exudes. Although the conjunctiva may be injected, swollen, and granular, it is not involved further in the pathologic process. The disease is caused by an organism belonging to the genus *oïdium*, the spores of the fungus finding entrance owing to the injury of the skin surface. The affection may be mistaken for epithelioma, tuberculosis, or syphilis, and is differentiated by the clinical appearances and by an examination of the secretion from the miliary abscesses which will reveal the organisms. From the ocular standpoint the disease has been well described in this country by Casey A. Wood, W. H. Wilder, and Edward Jackson.

Treatment.—This should consist of excision of the diseased areas, the application of the *x*-rays, and the internal administration of large doses of iodid of potassium. Ectropion may occur and may need a plastic operation for its relief (Wilder).

Hordeolum, or **stye,** consists of a localized, suppurating inflammation of the connective tissue in the margin of the lid or of one of the glands of the follicles of the cilia (Zeiss's glands), and is almost always due to staphylococcus infection. This may remain a tender, circumscribed swelling, which becomes invested with a yellow cap, indicating suppuration, or it may cause considerable pain, with edematous swelling of the entire lid and chemosis of the conjunctiva. It is known by the name *hordeolum externum*, to distinguish it from a *hordeolum internum*, which is the result of suppuration of a Meibomian gland. Some persons are subject to a mild type of styes which appear in the form of superficial pustules along the margin of the lid. A characteristic feature of hordeolum is its tendency to recur, and a single stye, or several at a time, may appear again and again for many weeks. Recurring hordeola may be the starting-points of a chronic blepharitis. Driving in the cold or dust and the strain of uncorrected ametropia predispose to this disorder. Frequent "attacks" of styes indicate derangement of health, and are especially associated with constipation and menstrual irregularities. Girls about the age of puberty are commonly affected.

Treatment.—A stye sometimes may be aborted by the vigorous application of a hot boric acid lotion or an ointment of the red or yellow oxid of mercury; the same end is obtained by painting the inflamed surface with collodion. In the event of failure, suppuration should be encouraged by repeated applications of small compresses steeped in hot water, and on the earliest appearance of pus a deep incision should be made through the base of the swelling, parallel to the edge of the lid. In persistent and recurring formation of styes treatment with bacterial vaccines controlled by determining the opsonic index has achieved excellent results.

Exanthematous eruptions on the eyelid are found during the course of the eruptive fevers. The pustules of small-pox, if they appear upon the eyelids, form by preference at the commissures, and in con-

nection with the follicles of the eyelashes. The subsequent pitting from loss of tissue may cause considerable disfigurement.

Sometimes a pustule declines to heal and forms a chronic *post-variolous ulcer*. *Vaccine vesicles* (vaccine blepharitis) may form on the lid-margins from accidental inoculation—*e. g.*, with the finger-nail previously in contact with a vaccine-pox or vaccine virus. The vesicles may develop into a severe ulcer, and the bulbar conjunctiva may be involved.

Eczema of the lids, independently of that variety which is located upon the ciliary margin and which is one of the forms of blepharitis, may appear upon the general cutaneous surface of these structures, usually in association with its presence elsewhere on the face and scalp, and is seen in the *erythematous*, *vesicular*, and *pustular* varieties.

Eczematous eruptions upon the lids are also associated with inflammations of the cornea and conjunctiva, and arise under the influence of prolonged bandaging. Atropin, when it produces conjunctivitis (see page 228), may cause an eczema of the lids and surrounding face.

Treatment.—This depends upon the character of the eruption. If this is vesicular, a useful application is a drying powder composed of starch, oxid of zinc, and camphor; if crusts have formed, these should be removed with as little bleeding as possible and with the aid of an alkaline solution, maceration of the epidermis being avoided, and one of the following ointments employed: Plain oxid of zinc, or equal parts of oxid of zinc and vaselin to which 20 grains (1.3 gm.) of calomel have been added; or subnitrate of bismuth in an ointment. Itching is relieved by the application of *lotio nigra* followed by zinc ointment. If the disease assumes a chronic type, some preparation of tar (*pix liquida* or *oil of cade*) may be used. Good results follow the use of aristol ointment, both in subacute and chronic cases.

As constitutional remedies, quinin, iron, and strychnin are recommended, and arsenic if the type is chronic. Proper regulation of diet, an occasional saline laxative, and good hygiene are important measures.

Herpes zoster ophthalmicus is a specific infectious, and possibly contagious, exanthem (Van Harlingen) characterized by an eruption of vesicles, situated upon inflamed bases, over the area supplied by two of the three branches of the ophthalmic, or first division of the trigeminus —viz., the frontal, through its supra-orbital and supratrochlear branches, and more rarely the nasal nerve.

Neuralgic pain, heat, and redness of the skin precede the vesicles, which, varying in size from a pin's head to a split pea, appear in distinct crops or coalesce in irregular patches. At first they contain a clear yellow fluid, later becoming turbid, until at the end of a week or more they dry up, and the brown scabs drop off, leaving beneath decided and often disfiguring scars.

The disease may be mistaken for erysipelas, from which it should be distinguished by the acute neuralgic pain and the formation of the vesicles in the course of a given set of nerves.

Serious involvement of the eye itself, by the formation of blebs upon

the cornea, and by inflammation of the iris and ciliary body, is often associated with the disorder. More or less conjunctivitis is always present. The blebs on the cornea rupture and form ulcers, which leave permanent scars, and the iritis and cyclitis may pass on to a destructive inflammation of the deeper coats of the eye (ophthalmitis). Atrophy of the optic nerves and paralysis of the oculomotor and of the superior oblique have followed ophthalmic herpes; a form of parenchymatous keratitis which precedes by several days the cutaneous lesions of herpes has been reported (Terrien). Acute glaucoma and glaucoma secondary to cyclitis have been observed.

Inflammation of the tissues of the eye is most apt to occur when the nasal branch is affected, and the vesicles extend to the tip of the nose, because from this branch, through the lenticular ganglion, arise the nerves supplying the iris, ciliary body, and choroid. This is not an invariable rule, and destructive disease of the eyeball may appear even when the nasal branch is not involved. A severe and most intractable neuralgia often remains after the subsidence of the eruption.

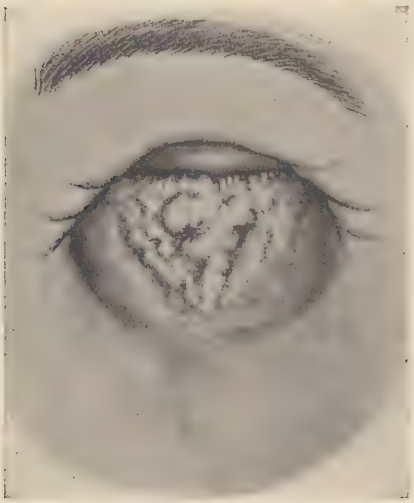


Fig. 84.—Extensive coalescing herpes confined to the lower lid.

Herpes zoster ophthalmicus is more frequently seen among elderly people of feeble nutrition than among adults and young children, but the latter may be attacked even in the absence of constitutional depression.

Treatment.—The disease runs an acute course and tends to spontaneous recovery in two or three weeks. Locally, anodynes are useful—lead-water and laudanum, weak carbolic acid lotions, and preparations of belladonna. Ichthyol ointment is valuable. Desiccating powders (rice-starch, calomel, zinc oxid) are useful. Severe pain must be mitigated by opiates and morphin hypodermically, while the best constitutional remedies are full doses of quinin and iron, and later arsenic. Salicylate of sodium and aspirin are valuable, and McNab recommends ionic medication, with sulphate of quinin by means of the positive pole over the affected area. The postneuralgic pain may be relieved by croton chloral hydrate in doses of 5 to 10 grains (0.324–6.5 gm.) every four hours, and by the use of a mild galvanic current. If conjunctivitis, keratitis, iritis, or cyclitis arises, it requires the treatment directed to the relief of such conditions, which is detailed in the special sections devoted to their consideration.

Herpes Facialis of the Lids.—Occasionally one or several groups of herpes vesicles develop upon the eyelids. The lesions usually appear

in the form of a small cluster or a coalescent patch. The lid is swollen, reddened, and the disease gives rise to a burning and itching sensation. The lower lid is affected more commonly than the upper.

Treatment.—The best application is ichthyol ointment, and under its influences the lesions rapidly disappear. The associated conjunctivitis should be treated with the usual applications—boric acid and argyrol.

Blepharitis is the term applied to the various grades of subacute and chronic inflammation of the border of the eyelid, which, for clinical purposes, may be gathered into two groups—*non-ulcerative* and *ulcerative blepharitis*. The former may be studied under several subdivisions:

1. **Hyperemia of the Lid-border** (*Hyperæmia Marginalis; Vasomotor Blepharitis*).—The margins of the lids have an unpleasant, slightly swollen, red appearance. Exposure to cold wind or any strain upon the accommodation causes a feeling of heat, followed by burning and lacrimation. The redness is caused by the passive congestion of the superficial blood-vessels. Scales or crusts are absent or but sparingly present.

2. **Simple Blepharitis** (*Seborrhea of the Lid-border; Blepharitis Ciliaris; Squamous Blepharitis*).—This variety depends upon an abnormal secretion of the sebaceous glands, and results in the formation of scales and crusts situated on the margin of the lids at the bases of the eyelashes, or adhering to them, and may appear in either a dry or a moist form. Removal of the hardened sebum exposes the skin, shining, red, and occasionally abraded. There is usually slight conjunctivitis. An accompanying seborrhea of the eyebrows and scalp may be present; both lids are invariably affected, and the patients complain of burning, inability to perform close work, and some dread of light.

Exposure to cold and dust and the use of the eyes quickly increase the congestion of the lids. If the disease is of long duration or is subject to frequent relapses, considerable thickening of the lid-margins is evident, due to the inflammation surrounding the glands in the skin and tarsus. Crust formation on the lid-margins due to a deposit of dried conjunctival secretion may simulate blepharitis. In these circumstances removal of the scales will demonstrate that the underlying skin is normal.

The second, or *ulcerative*, form of blepharitis appears in several grades of severity as a special localization of—

Eczema Upon the Lid-border (*Blepharitis Ciliaris; Blepharitis Ulcerosa; Psorophthalmia; Lippitudo Ulcerosa; Tinea Tarsi; Sycosis Tarsi; Ophthalmia Tarsi, etc.*).

(*a*) *Superficial Form* (*Marginal Eczema*).—This resembles in general that variety which has been described as hyperemia of the ciliary margin. The patient suffers from "weak eyes" and from frequent attacks of redness and soreness of the borders of the lids, associated with the formation of crusts, small pustules, and ulcers at the roots of the lashes, without, however, seriously interfering with their nutrition or growth.

(*b*) *Solitary Form* (*Blepharo-adenitis Ciliaris, a name given by Arlt*).—This is characterized by the appearance of a circumscribed area of

thickening and redness of the lid-margin, upon which the cilia are matted together at their bases by the formation of thick yellow crusts. A single tuft of this kind may be present, or several on one lid-border; the process is frequently unilateral, in this respect being unlike the squamous forms, which are bilateral. Removal of the crusts evacuates a few drops of thin pus from the surface of the ulcer which lies beneath, and the cilia, which usually come away with the scab, have swollen and thickened roots. Spots of eczema at the nares and in the hair of the scalp may be present at the same time, as well as disease of the lacrimal passages.

(*c*) *Pustular Form* (*Blepharitis Ciliaris Ulcerosa*).—This manifests itself as an eczema of the lid-margins, in its worst types involving the four ciliary borders. Thick yellow crusts, which mat the eyelashes, form along the palpebral margins, covering deep ulcers which readily bleed, and which, often crater shaped, pass inward to the tarsus.

The inflammatory process, if unchecked, seriously interferes with the nutrition of the lashes and the edges of the eyelid. The former become stunted, curled, misplaced (*trichiasis*), or drop out, and may be entirely absent (*madarosis*, *tylosis*). The latter assume a rounded shape, are swollen, reddened, thickened, slightly everted, and deprived of cilia (*lippitudo,* or "blear eye," *hypertrophic blepharitis*), and if the punctum lachrymale is displaced or closed, an overflow of tears adds to the discomfort of the patient.

It is not always possible thus sharply to separate the various types of blepharitis, as they often shade one into the other; nor is it always safe to decide between those which arise from glandular hypersecretion and those which are due to eczema. After the cure of an ulcerative variety, small scales may form resembling the simple or squamous type, while the latter may also lead to, or be associated with, ulcerations.

Terson suggests a classification of affections of the lid-margins from the dermatologic standpoint. He would distinguish two main groups, the suppurative and the squamous. The former, for the most part, includes affections of the hair-follicles and of the surrounding tissue—that is to say, either a *folliculitis* or a *parafolliculitis*. He regards ulcerative blepharitis as a process analogous to sycosis. The squamous form of blepharitis he classifies with seborrhea.

Etiology.—In the majority of instances blepharitis is a disease of childhood, and is common near the age of puberty; the aggravated forms, especially those resulting in chronic changes in the ciliary margins, are frequently seen in adults as the result of neglect. The malady may follow in the wake of an exanthem, particularly measles, and finds many subjects among anemic children of strumous or tuberculous habit, with blond hair and pale complexion. The usual presence of ametropia has led to the belief that this causes blepharitis (Roosa). There is no doubt that it aggravates and fosters the condition. In some families blepharitis is hereditary (Fuchs).

Of considerable importance in the origin of this affection are inflammations of the tear-sac, stricture of the nasal duct, and obstructive

disease of the posterior nares, although it may be difficult, in individual cases, to decide whether the blepharitis has caused the closure of the lacrimal passages, or whether this has developed the blepharitis. Blepharitis is aggravated and excited by exposure to wind, dust, and heat, and by intemperate habits and loss of sleep. Finally, some instances appear to arise from an abnormal and probably congenital shortness of the lids (*microblepharon*), resulting in their insufficient closure during sleep (Fuchs).

Staphylococci are found in the pustules. McNab has frequently discovered the Morax-Axenfeld bacillus in some varieties of marginal blepharitis. Stubborn varieties may depend upon eczema seborrhoicum of the face; rarely the trichophyton fungus is found (*blepharitis trichophytica* of Mibelli). According to Raehlmann, the Demodex folliculorum may cause the disease (*blepharitis acaria*). It is, however, a not uncommon inhabitant of the normal eyelid. *Favus*, in the form of dirty, yellowish-white crusts, occasionally appears upon the eyelids, and may be mistaken for blepharitis. Microscopic examination of the crusts would reveal the mycelium and the conidia. The invasion of the follicles of the eyelashes by the *Trichophyton tonsurans*, when the brow or beard is similarly affected, produces an appearance closely resembling severe blepharitis.

Treatment.—This differs with the type of the disease, but in all cases the refraction of the eye should be ascertained and any anomalous condition corrected with suitable glasses. This will often cure an ordinary hyperemia of the lid-margin, but if it is not sufficient, in addition to soothing lotions, the daily use of a douche of water at a temperature of 68° F., to which is added a little *eau de cologne* or alcohol, is serviceable. Stimulating salves do not yield good results in this variety, but the edges of the lids may be anointed with almond oil or vaselin.

In the cases classified among the seborrheas all crusts and scales should be removed by means of alkaline solutions—bicarbonate or biborate of soda, gr. viij to f℥j (0.52 gm. to 30 c.c.)—or with a 5 per cent. solution of chloral (Gradle), and one of the following ointments applied once or twice daily: yellow oxid of mercury, gr. j to ℥j (0.065–to 3.885 gm.), zinc ointment, or the salve advised by Gradle milk of (sulphur and resorcin, 3 per cent.).

Great care must be exercised to remove the crusts from all the ulcerated varieties, either with the lotions which have been mentioned or, after softening, with forceps, before the application of any salve. A satisfactory method is daily gentle scrubbing of the lid-margins with the lather of a good neutral soap. For this purpose special soaps (ophthalmic soaps) may be employed. Red or yellow oxid of mercury or dilute citrine ointment or ichthyol (2–10 per cent. of the ammoniacal salt) may be applied to the lid margins.

In chronic cases all loose cilia should be extracted with epilating forceps, and any deep ulcers should be touched with the point of a crayon of nitrate of silver, or penciled with a solution of the same drug, or treated with a mixture of corrosive sublimate in glycerin (1 : 100 to 1 : 30

—Despagnet). In severe forms, or when it is desirable to try other remedies, diachylon ointment, 15 to 240 grains (0.972–15.5 gm.) of vaselin, boric acid ointment, 10 to 100 grains (0.65–6.5 gm.), or aristol ointment, 15 to 150 grains (0.972–9.72 gm.), will be found useful. Fridenberg recommends expression of the lid-margins in order to remove the pathologic secretion from the glands and ducts. Picric acid (0.8–1 per cent.) in glycerin (Fage), and sulphate of zinc, gr. ij to f℥j (0.13 gm. to 30 c.c.), if the Morax-Axenfeld bacillus is present (McNab), have been advised. Stubborn blepharitis has been successfully treated by *ionic medication*, after the manner of Wirtz. A ½ per cent. solution of sulphate of zinc is employed; special electrodes are required.[1]

If the lacrimal passages are obstructed, they must be rendered patulous, and in all cases the anterior and posterior nares should be explored for disease.

The constitutional remedies include iron, quinin, and, if struma is present, cod-liver oil and lactophosphate of lime, with iodid of iron or syrup of hydriodic acid.

Blepharitis may be a mild affection and yield readily to treatment; or it may be stubborn, and require constant attention and frequent change in local measures to prevent deformities in the lid-margins.

Phthiriasis (**blepharitis pediculosa**) occurs when the pediculus pubis or crab-louse forsakes its seat of predilection and finds a habitat among the eyelashes. The cilia appear sprinkled with a fine dark powder—the eggs of the parasites—which are usually found partially buried, head foremost, in the hair-follicles. There are some itching and redness. The affection in most instances has been observed in children. The lice may be removed by the application of blue ointment or a careful penciling with a strong bichlorid solution.

Sporotrichosis of the Eyelids.—This disease may attack the eyelids in the form of a dermic granuloma with swelling of the preauricular, submaxillary, and cervical lymphatic glands (H. Gifford, Morax). Ulceration of the lid border and small abscesses of the lid skin may arise. Syphilis, tuberculosis, and blastomycosis must be excluded by cultivating the germ on maltose agar. The *Sporothrix Beurmannii* is the fungus which usually is the active agent.

The *treatment* consists in local antisepsis and the administration of iodid of potassium.

Syphilis of the Eyelids.—Syphilitic affections of the eyelids exist either as the primary sore or as secondary or hereditary manifestations. A chancre usually appears on the area included by the lid-borders and inner canthus, the tarsal conjunctiva and the culdesac (deBeck). The lesion, generally on one lid, but in rare instances bilateral, begins as a pimple, which gradually develops into a characteristic, somewhat saucer-shaped ulceration, with rather rounded edges and indurated base. The lymph-glands in front of the ear and at the angle of the jaw are enlarged. Contagion has often occurred by the application of the lips

[1] For details consult Klin. Monatsbl. f. Augenheilk., Nov.–Dec., 1908; also Ophthalmoscope, Jan., 1911, p. 18.

or tongue of an individual suffering from mucous patches in the mouth—as, for instance, in the act of kissing; or by the filthy practice of attempting to remove a foreign body with the tip of the tongue. Soiled fingers have also carried the contagion.

It is possible to mistake the affection for a stye, suppurating chalazion, ulcerated tear-sac, vaccine ulcer, or small rodent ulcer. In doubtful lesions a search for the *Spirochæta pallida* should be made; the Wassermann test should be applied.

Treatment.—Locally, the ulcer may be dressed with black or yellow wash. As soon as the diagnosis is established, the ordinary antisyphilitic remedies should be exhibited, especially salvarsan or neosalvarsan.

The lesions of *secondary syphilis* upon the eyelids require no special description.

Among the later manifestations *gummas* of the skin of the lid, which break down into ulcers,—so-called *tertiary ulcers*,—are described.

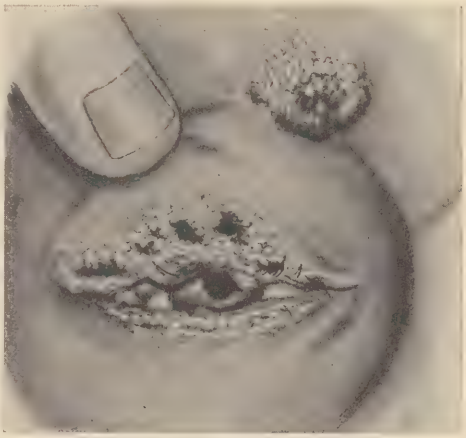


Fig. 85.—Extensive gumma of the upper and lower lid of the right eye.

A papular eruption may appear upon the eyelids of children the subjects of hereditary syphilis shortly after birth. A form of blepharitis, characterized by sharply ulcerated spots, has been described as the result of hereditary syphilis, and in subjects of this dyscrasia absence and falling out of the eyelashes have been seen. The latter condition also arises during secondary syphilis.

Tumors and Hypertrophies.—A variety of growths, cystic and solid, are found upon the eyelid and its border. Along the latter, *warts* or *papillomas* are common. These are benign, except when in elderly persons, through irritation, they may take on an epitheliomatous nature. They should be cut off and their bases cauterized. In place of the ordinary elevated wart (*verruca*) a flat variety of the growth, which in elderly persons may be pigmented, often develops (*keratosis senilis pigmentosus*). Superficial wart-like processes situated upon the intermarginal area of the lids have been described by Birch-Hirschfeld (*acanthosis nigricans*).

Small clear *cysts*, arising from Moll's glands, are common along the ciliary margin, often giving rise to considerable irritation. They should be punctured.

A reddish, wart-like mass may occur at the mouth of a Meibomian gland-duct. This is to be treated like an ordinary wart.

Angiomas (*nevi*) are usually congenital growths, and exist either as bright-red spots (*capillary angiomas*) or in the form of elevated, bluish,

somewhat lobulated, *cavernous* growths, which may assume large proportions and extend from the lid to the forehead and temple. These cavernomas become turgid, purplish in color, and apparently increase in size if the child cries. They should be dealt with early in their existence, lest they spread into the orbit. In a patient under the author's care a tumor of this character extended to the apex of the orbit and involved the lacrimal gland. A tumor occupying the upper surface of the tarsus, soft in consistence and bluish in color, has been noted in the eyelids of babies. Clinically, it may be mistaken for a cavernoma, but examination after removal, which is usually accomplished without difficulty by an ordinary dissection, shows, as in Arnold Knapp's specimen, that it is composed of open spaces containing blood, which are lined with large endothelial cells, similar cells occupying the intervening regions. Angiomas arising from the lymphatics are known as *lymphangiomas;* those which develop from the blood-vessels, as *hemangiomas.* Nevus pigmentosus, and moreover, as the starting-point of a malignant growth, has been observed on the eyelids.

That operative interference should be practised which promises the least subsequent deformity to the lid. When small, capillary angiomas may be excised or cauterized with nitric acid; if of a larger variety, their blood-vessel structure may be destroyed with galvanocautery needles; or *electrolysis* may be tried, three gold-plated needles attached to the negative pole being inserted in various positions in the nevoid tissue, while the positive pole is attached to some distant point—for example, the arm. The *seance* should last from ten minutes to half an hour, according to circumstances. The application of carbon-dioxid snow has been efficient in the treatment of some angiomas. Radium has also been tried. It is possible sometimes to excise large cavernous angiomas, and if there is not sufficient skin to cover the defect immediately, to accomplish this subsequently by skin-grafting. Indeed, excision by an ordinary dissection, if it can be accomplished without serious loss of tissue, is a desirable method of treatment. In infants, however, the danger of shock and hemorrhage is great.

Occasionally ulceration occurs in an angioma and is followed by serious hemorrhage.

A disease characterized by an increase in the volume of the skin of the lid, which becomes folded and falls over its margin, but appears atrophic and may be transiently red, like the color of the cheek, has been described by Rohmer and others, and to it the name *angiomegaly* has been given. It symmetrically affects the upper lids, and has been attributed to a structural or functional anomaly of the vascular system.

Cutaneous horns (*fibroma; molluscum fibrosum*) occur as connective-tissue new growths, either sessile or pedunculated, sometimes associated with numerous similar tumors elsewhere on the body. A *cornu cutaneum* may grow from the margin of the lid (Fig. 86).

Neuromas of the plexiform variety occur on the eyelids, and *neurofibromatosis* accompanied by optic neuritis has been observed, and with hydrophthalmos on the corresponding side. *Lipomas*, which are prob-

ably extensions from the orbit, are benign growths which may be removed by careful dissection. *Ptosis lipomatosis*, at one time attributed to an accumulation of fat in the connective tissue of the upper lid, causing it to droop and its covering fold to hang over the palpebral border, is due to relaxation of the fascia connecting the skin with the tendon of the levator. *Fat hernias* of the upper lid have been described by Schmidt-Rimpler as the result of a congenital extension of the orbital fat through a defect in the orbicularis muscle.

Rare forms of benign tumors are *adenoma* of the sweat-glands and their follicles, *adenoma* of the Meibomian glands (Knapp), *papilloma* of the ciliary border, *enchondroma* of the tarsus, and *myoma* of the orbicularis (Schnaudigel). Hypertrophy and ossification of the tarsus have been reported (Herbert).

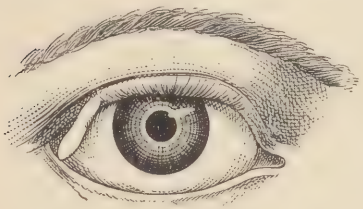


Fig. 86.—Cornu cutaneum of the upper eyelid (from a patient in the Jefferson Medical College Hospital).

Fig. 87.—Neuroma of the right upper eyelid and adjacent temporal region (from a patient in the Philadelphia General Hospital).

Xanthelasma (*xanthoma*) is a connective-tissue new growth, with fatty degeneration, usually seen in the form of narrow, semicircular patches, most common upon the upper eyelids, although all four lids may be affected. The patches are yellow or buff colored, and on a level with the surrounding skin or slightly raised above it.

Excision, if this may be performed without producing ectropion, is the simplest method of treatment, but often yields unsatisfactory results, inasmuch as the xanthelasma reappears in the region of the excision. Electrolysis has also been recommended and often produces good results. High-frequency currents applied to the plaques by means of a special electrode are advised by Bordier. The application of trichloracetic acid to small xanthelasma patches has been commended, and recently the application of radium has been recommended by Schindler.

Chalazion (*Meibomian Cyst; Tarsal Tumor*).—This is a small tumor due to a chronic inflammation of a Meibomian gland and the tissue which surrounds it. The growth begins by retention of the secretion of the Meibomian gland, followed by a peri-adenitis and destruction of the tarsal cartilage, with passage of the tumor toward the conjunctiva

(*internal chalazion*) or to the skin (*external chalazion*). Usually the process is a *chronic* one; sometimes it assumes an *acute* nature, and there is inflammatory reaction (see also *hordeolum internum*). A chalazion may form in the excretory duct of a Meibomian gland, and then projects in a nipple-like body from the edge of the lid. Chalazia may be single or multiple, and in severe cases recurrences may be frequent until a chronic infection of the Meibomian glands and alteration of the tarsal cartilages take place. To this condition Weymann has given the name *tarsadenitis Meibomica*. In association with nasal ozena an affection of the Meibomian glands has been described in which they become chronically inflamed, and pus, containing the ozena bacillus, exudes from their ducts.

Cause.—The cause of chalazia is not known, although Deyl and Hála maintain that they represent an infectious, bacterial process, the active bacilli being identical with xerosis bacilli. They may be associated with inflammation of the border of the lid and stoppage of the duct of the gland. Individuals affected with these growths not infrequently have ametropic eyes, especially when there is a tendency to recurrence in crops, like styes. They are more common in adolescence than in youth, childhood, or in old age.

Symptoms.—The tumor grows slowly, unless it is of the acute type, and forms a firm swelling attached to the tarsus. The skin usually is freely movable over it; on the conjunctival surface a discolored patch marks its position. Suppuration may take place in the growth.

A so-called acute chalazion may be mistaken for an external stye, from which it is to be distinguished by the more circumscribed character of the inflammation, and by the fact that the stye points in the edge of the lid; and a chronic chalazion for a sebaceous cyst from which it may be differentiated by the firmness of its attachment to the tarsus. A chalazion, a small sarcoma of the lid, and even a beginning glandular carcinoma have been confounded.

Pathologic Anatomy.—A microscopic examination reveals a collection of cells, the majority of which are of the small round variety, having their origin in the acini of the Meibomian glands. Sometimes large multinuclear (giant-) cells are evident, though inoculation experiments have shown that these are not tuberculous in type. The central part of the growth later undergoes a mucoid or colloid degeneration, and a cavity appears, which is filled with a cloudy fluid. There is no true capsule, and there are consequently no characteristics of a true cyst.

Treatment.—An ointment—2 grains (0.13 gm.) yellow oxid of mercury to 1 dram (3.9 gm.) of vaselin or lanolin—persistently rubbed into the skin over a chalazion will occasionally cause it to disappear, but usually it is necessary to remove it, according to the method described on page 599. The eyes of patients who suffer from chalazia are usually ametropic and suitable glasses should be adjusted.

The malignant growths which appear upon the eyelids are *sarcoma, cylindroma, carcinoma* in the form of *epithelioma* or of *rodent ulcer,* and *lupus.*

Sarcoma occurs as a primary tumor in both upper and lower lids, about 80 cases being on record, and usually is seen in children. The recorded cases have been analyzed by Wilmer, Veasey, Friedenwald, Shumway, and the author. According to Veasey, the youngest subject of lid sarcoma was seven months old and the oldest seventy-six years. At first the growth is slightly elastic, and the skin moves over it freely, but the tendency is to rapid growth, ulceration, and involvement of the orbit. The tumor may attain large proportions. In a patient under the care of J. Chalmers Da Costa and the author the weight of the

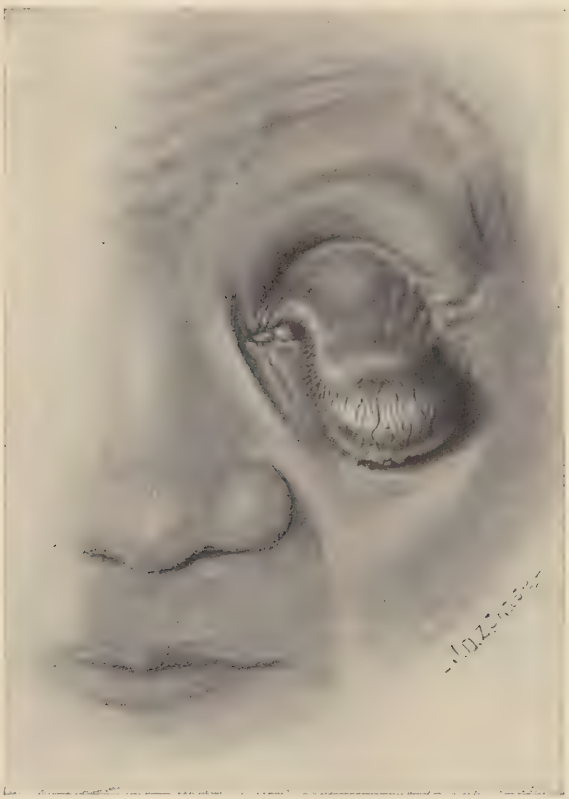


Fig. 88.—Sarcoma of lid (from a patient in the University Hospital).

neoplasm was 247 grams. The various types of sarcoma (myxosarcoma being frequent) have been seen in this region, both pigmented and non-pigmented, and the tumor has been known to follow a contusion. Cylindroma, somewhat allied to sarcoma, but less malignant, composed, microscopically, of hyaline cylinders, occurs at times in the eyelids of adults, and is of slow growth.

An early removal of the tumor is urgently indicated, but in spite of operation there may be recurrence or metastasis. Radium has been successful in some instances (Callan, Abbe).

Lymphomas occur in the lids and orbits in patients suffering from leukemia. They are often symmetric. They cannot be distinguished histologically from round-celled sarcomas. Lymphoma of the lower lid may be part of a general lymphomatosis (Coats).

Carcinoma of the eyelid often appears in the form of *rodent ulcer* (Jacob's ulcer), which is a type of epithelial cancer, being, according to F. H. Montgomery, practically a superficial carcinoma of the tubular variety. It is characterized by slow ulceration and non-involvement of the neighboring lymph-glands, and is usually seen in elderly persons.

The growth begins as a pimple, over which a crust appears. Gradually an ulcer forms, which slowly spreads with indurated and elevated

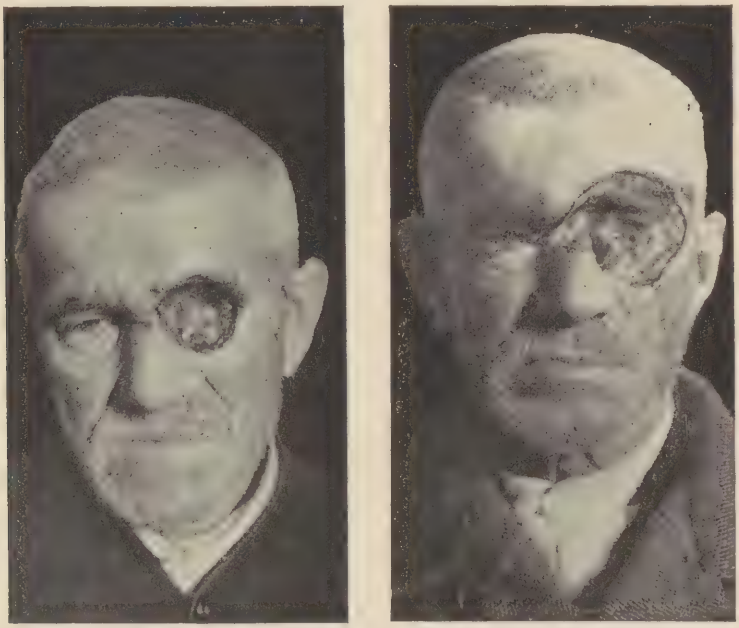


Fig. 89.—Destruction of eyeball and orbital tissues by a rodent ulcer: five years between the two stages (from a patient in the Philadelphia General Hospital).

edges, and, if unchecked, involves all the tissues and destroys the eyeball. Often many years elapse before the ulcer attains any considerable size. The most common point of origin is the inner end of the lower lid.

The slow growth and absence of lymphatic involvement, together with the age of the patient, suffice to distinguish rodent ulcer from a tertiary syphilitic sore.

It may be confounded with lupus, but the latter occurs in younger subjects, is more inflamed and less indurated, the ulcerations proceed from many points, and are generally associated with lupus elsewhere in the body.

Epithelioma with the ordinary clinical characteristics may attack the eyelid, and is one of the commonest tumors of this region. It usually

begins at the lid margin, and is more frequent on the lower than on the upper lid. It not infrequently is situated at the outer commissure and involves both lids (Fig. 90). Microscopically, it consists chiefly of a downgrowth of the interpapillary processes of the rete. The epithelial plugs often contain "cell-nests." According to Ginsberg, a certain number of growths recorded as epitheliomas are really *endotheliomas* (Fig. 91). *Glandular carcinoma*, having its point of origin either in the Meibomian or in Krause's glands, may also occur in this region.

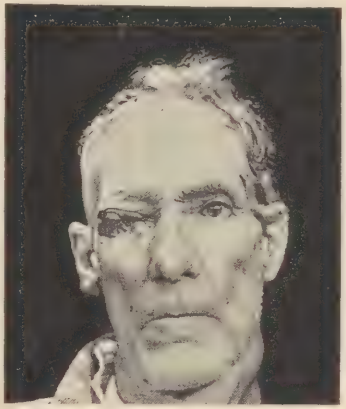


Fig. 90.—Epithelioma of the eyelid (from a patient in the Jefferson Medical College Hospital).

Treatment.—Certain local remedies, as aristol, chlorate of potassium, and injections of pyoktanin, have been recommended. If the disease is advanced, Canquoin's paste, chloracetic acid, scraping, and the actual cautery have been employed to check the ulceration.

In recent years great advances in the treatment of the various malignant growths of the eyelids have been made, especially in the application of the *x*-rays.

Fig. 91.—Epithelioma of eyelid encroaching on eyeball (from a patient in the Philadelphia General Hospital).

At the author's request Dr. Henry Pancoast, Professor of Röntgenology in the University of Pennsylvania, has prepared the following directions, which will be found useful:

An important consideration in *x*-ray applications in the neighborhood of the orbit is the possible injury to the eye that may result from the exposures. Ulceration and opacity of the cornea, severe conjunc-

tivitis, edema of the conjunctiva and lids, and optic neuritis have been noted. The eye should always be carefully protected.

Epitheliomas involving the lids are frequently troublesome and obstinate, because there is a tendency to allow too little exposure to the rays lest the eye be damaged, or sufficient treatment cannot be given on account of the danger of injuring the ocular coats. If the growth overlies the eyeball, cocain may be introduced, and then a hard-rubber, ivory, or metal eye spatula may be inserted under the lid. All the surrounding healthy parts should be covered by an impenetrable protective. The quality and quantity of rays used are most important. The tube should be soft, such a one which has a resistance equal to 1 or $1\frac{1}{2}$ inches of spark-gap, and it should be placed as near to the area as possible. A current of 1 to 3 milliamperes may be used in the secondary. With the aid of a mechanical spring-interrupter the average duration of each exposure should be from five to ten minutes. On account of the latency of *x*-ray effects many operators advise giving the exposures in series. Four or more applications are made on successive days, and then an interval of several days follows before the next set of exposures is given. With such a technic the necessary number of applications does not entail much danger to the eye. At the same time it should be borne in mind that preliminary partial or complete excision should be performed when it is not inconsistent with good cosmetic results.

Although, according to Pancoast, excellent results have been obtained in many instances in the treatment of epitheliomas of the eyelids by the use of Röntgen rays, the results from radium he believes are preferable and more certain, and the latter agent can usually be employed to better advantage and with greater safety to the eye. The reaction to radium in the unprotected bulbar conjunctiva is very severe and the eye should, therefore, be adequately protected against the less penetrating beta rays. This can readily be accomplished when the application is made near the palpebral margin by inserting a piece of metal such as a Snellen clamp between the lid and the eye, after cocainization. Such treatment results in a minimum loss of tissue, and any resulting deformity can be corrected subsequently if necessary. Papillomas may be removed in the same manner.

Lupus vulgaris is a cellular new growth composed of variously shaped, reddish tubercles, which usually terminate in ulceration and extensive cicatrization. As this disease commonly appears on the face, it may also involve the eyelids.

The process begins in youth, often before puberty, and is slow in its course. The ulcers are apt to start from a number of points which coalesce; their edges are soft, and the discharge is offensive. *Syphilitic ulcers*, on the other hand, are deeper, more excavated, with harder margins, and their course is more rapid. *Tuberculosis of the lid* and *tarsus* is usually secondary to conjunctival tuberculosis; rarely it is primary and may simulate the clinical picture of chalazion (von Hippel).

Treatment.—Local application of caustic paste, erasion with a

curet and the actual cautery have been employed, and injections of tuberculin have been recommended.

Lepra.—Leprosy attacks the eyelids very frequently. According to Lopez, two-thirds of those affected with this disease suffer from lesions in this region. These consist of anesthetic patches of color slightly different from that of the surrounding integument, tubercles, loss of the eyelashes and eyebrows, and ectropion and entropion, the former occurring with extraordinary frequency.

Xeroderma Pigmentosum.—According to Greeff, the ocular affections in this disease are found both on the skin of the lids and the mucous membrane of the eye; more rarely on the cornea. The earliest evidences of the disease appear on the face, and particularly on the lids. After certain irritative symptoms, an atrophic process develops with areas of pigmentation, and even in the early stage of the disease the cilia fall out and disappear. Later, elevations of a warty appearance develop, the epithelial processes of which extend inward and become true carcinomas. The disease may occur in childhood as well as in adult age, and it is interesting that even in youth carcinoma may develop. L. W. Dean has reported favorable results from the use of cocoa butter locally and injections of autoserum. Thorium has been tried. The carcinomatous tumors may be removed if they are not too numerous.

Fig. 92.—From a photograph of a patient with syphilitic tarsitis under the care of Dr. Randall, in the Children's Hospital.

Elephantiasis arabum, a chronic hypertrophic disease of the skin and subcutaneous tissue, has appeared in the upper eyelid in consequence of an injury, but may also be congenital. According to Cirincione, a distinguishing feature of true elephantiasis of the lids is that at least two lids are involved, and generally four of them. Repeated attacks of erysipelas have etiologic importance. *Elephantiasis telangiectodes*, or that disease which consists in a hypertrophy of the skin and connective tissue, together with fatty tissue and distended vessels, occurs in the upper eyelid as a congenital affection.

Tarsitis, or inflammation of the tarsus, is usually syphilitic in origin, and presents great thickening of the tarsus, owing to diffuse gummatous infiltrations (Fig. 92). It may also be due to tuberculosis and to trachoma. As a rule, it is chronic in course; an acute form has been described. The disease may resemble a chronic marginal blepharitis, with the formation of crusts and ulcers at the mouths of the hair-follicles, but differs from the latter condition by the presence of considerable thickening and induration of the tarsus. Alteration of the tarsus, owing to chronic infection of the Meibomian glands, may arise, and has been referred to. Suppurative tarsitis has occurred;

the author has recorded one case of this character apparently due to influenza. An ulcerative variety due to syphilis has been described (Morax).

Treatment.—If syphilitic, tarsitis is amenable to the ordinary remedies; if not, much the same treatment described in connection with chronic blepharitis is applicable, especially the use of resolvent ointments.

Blepharospasm, or an involuntary contraction of a portion or the whole of the orbicularis palpebrarum, appears as either a *clonic* or a *tonic* cramp.

The former variety may consist merely in a twitching of a few fibers of the muscle, most commonly in the lower lid, very annoying, and often the cause of undue alarm. It arises from the strain of ametropia, prolonged eye use, and deficient amplitude of accommodation. It also occurs in a severe and intractable form, and occasions much discomfort and conjunctival irritation.

The *treatment* comprises the prescription of glasses and a general tonic. In stubborn cases fluidextract of gelsemium will occasionally afford relief. Conium internally, and the extract locally, have been recommended. In recent times hypodermic injections of 80 per cent. alcohol at the emergence of the facial nerve have been tried and satisfactory results have been reported.

Children are often affected, especially during their early school years, with undue winking of the eyelids, associated at times with jerky movements of the facial and other muscles. This form of nervous disorder was designated by Weir Mitchell *habit chorea*.[1] Almost invariably, blepharitis, follicular and phlyctenular conjunctivitis, and errors of refraction and heterophoria are exciting causes. Long-continued blepharospasm, especially in children with phlyctenular conjunctivitis, may give rise to *lid-edema*, due to pressure of the contracted orbicularis on the palpebral veins. Suitable glasses and appropriate local remedies, together with the exhibition of Fowler's solution, will usually bring about a cure.

Tonic cramp of the orbicularis follows the introduction of foreign bodies into the eye, the presence of inflammations of the cornea and conjunctiva, and fissures at the angles of the lids, and depends upon irritation of the peripheral trigeminal filaments.

More rarely a persistent lid cramp occurs, without obvious cause, and is unrelieved for weeks and even months. When the eyes are finally opened, there may be temporary blindness, without corresponding ophthalmoscopic changes; or permanent loss of vision, with gross lesions in the eye-ground.

The *treatment* demands the removal of any peripherally exciting cause—fissure, foreign bodies, phlyctenules, etc. Hypodermic injections of morphin have been used to control the trigeminal irritation, and in severe cases section of the supra-orbital nerve has been performed. Conium and gelsemium in the form of the fluidextract may be tried.

[1] Gowers gives the name "habit spasm" to the same affection.

They should be pushed to the point of tolerance. The hypodermic use of alcohol has been referred to.

Ptosis (*blepharoptosis*) is that condition in which the upper lid droops entirely or partially over the eyeball, and cannot be voluntarily raised. It is either congenital (see page 160) or acquired by reason of the development of fatty or other accumulations in the connective tissue of the lid (*pseudoptosis*, see page 171), or it arises from paralysis of the oculomotor nerve, and in rare instances from lesion of its cortical center (*paralytic ptosis*). Ptosis also occurs as the result of injury of the levator. In some cases of unilateral congenital ptosis, usually on the left side, while the eyelid cannot be voluntarily raised, it is elevated when the jaw is moved during eating (contraction of the levator in association with the external pterygoid) (see also Ocular Palsies).

Fig. 93.—Ptosis with edema of tissues, the result of laceration of the lid.

It is convenient to make reference in this place to other associations in muscle action. Thus, there may be contraction of the orbicularis with movements of the jaw, contraction of the levator with abduction and adduction, and, as reported by Zentmayer, contraction of the frontalis with abduction.

Treatment.—The medicinal treatment of ptosis calls for the exhibition of those remedies which control the supposed cause of the palsy—mercury and iodids in syphilis, salicylic acid in rheumatism.

The surgical treatment will be found on page 600.

Blepharochalasis, or relaxation of the skin of the lid, due to atrophy of the intercellular tissue, has been described by Fuchs and other writers. The skin of the lid is thin, much wrinkled, and its superficial

veins are dilated. The condition may be remedied by excising appropriate portions of the relaxed tissue and uniting the cut edges with sutures.

Lagophthalmos, or an inability to close the eyelids, is either paralytic or non-paralytic, and usually results from paralysis of the facial nerve, as in Bell's palsy, but also occurs in tumors of the orbit, exophthalmic goiter, and staphyloma. The highest grade of lagophthalmos appears as a congenital defect (see page 158). *Paralysis of the orbicularis* has been described as a hysteric phenomenon, but it is doubtful if in these circumstances it really has been observed. Orbicularis palsy may follow an injury, and the author has seen bilateral paralysis of the orbicularis after fracture of the base of the skull, with only slight involvement of the lower facial area. Failure of the lids to come in contact with the globe at the outer canthus has been recorded as a congenital condition.

The chief danger of the affection is ulceration of the cornea from exposure, rendered all the more certain should disease of the trigeminus also exist.

Treatment.—In paralytic lagophthalmos the primary cause of the affection must be treated: in the non-paralytic varieties, and in any form in which the vitality of the cornea is threatened by its exposure, the operation of tarsorrhaphy may be employed (see page 603).

Symblepharon,[1] or a cohesion between the eyelid and the ball, may be complete or partial, acquired or congenital (see page 158). The most usual causes are injuries, especially burns with acids or lime. Symblepharon also follows diphtheritic conjunctivitis, trachoma, pemphigus, and occasionally purulent conjunctivitis; but the shortening of the conjunctival sulcus, which occurs by a species of drying of the conjunctiva, presently to be described, must not be confounded with a true symblepharon. The attachment may be merely slight bands between the conjunctival surface of the lid and ball, or, in the more complete cases, the cornea may also be involved in the cicatricial union, and vision be seriously disturbed. The lower lid is most usually involved in the process; the upper may also participate (Fig. 94).

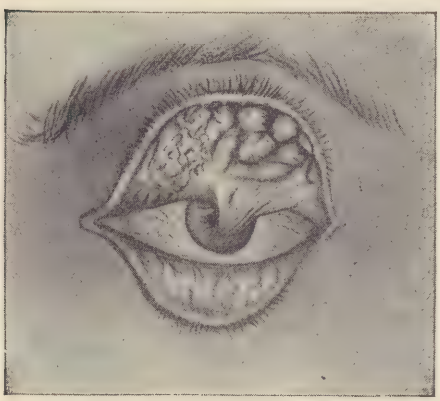


Fig. 94.—Symblepharon, the sequel of purulent conjunctivitis (from a patient in the Philadelphia General Hospital).

Ankyloblepharon, or that condition in which the borders of the

[1] Symblepharon really belongs to diseases of the conjunctiva, but is conveniently inserted in this place.

two lids have grown together, may be congenital or acquired, and, like the preceding affection, partial or complete.

The same causes which originate symblepharon are here active, and varieties are described in which the union takes place not by a growing together of the lids, but by the organization of a membrane, the result of croupous conjunctivitis.

Blepharophimosis is the name given to that condition which arises through a contraction of the outer commissure of the lids, and results in shortening of the palpebral fissure.

It is commonly seen in cases of long-standing conjunctivitis with irritating secretions; for instance, in chronic conjunctivitis and in some of the forms of granular lids.

Treatment.—After an injury, or during the course of a local disease, likely to result in one of these complications, scrupulous care must be exercised to avoid it. The formation of granulation tissue may be broken up with a probe, and it has been advised to place a piece of gold-beater's skin or the thin skin from the inner surface of an egg-shell (Coover) between the lid and the ball to prevent adhesions.

The surgical treatment of these affections is described on page 604.

Trichiasis; Distichiasis.—*Trichiasis* is that affection in which the lashes are misplaced and turn inward against the eyeball; *distichiasis* is that condition in which incurved rows of supplementary cilia are developed from the intermarginal part, close to the opening of the tarsal glands.

The most usual causes of trichiasis are chronic inflammations of the lid-borders—blepharitis and granular conjunctivitis.[1] Distichiasis, in rare instances, is congenital, or develops about the age of puberty. The cilia rubbing against the cornea produce constant irritation, and may lead to ulceration.

Treatment.—If not too numerous, the lashes having a faulty direction should be removed with cilium forceps, and when they grow again, the procedure repeated; their reappearance may sometimes be prevented by destruction of the hair-follicles by galvanopuncture. Other operations consist of strangulation of the roots of the incurved lashes by a subcutaneous ligature, excision, and the various modifications of single and double transplantation of the entire ciliary border (see chapter on Operations).

Alopecia of the eyelids, the loss of the lashes depending upon the fact that the patient, usually a hysteric girl, systematically pulls out the cilia, has been described by H. Gifford. The author has seen several cases. Sudden *turning gray of the eyelashes* has been recorded by Hirschberg after phlyctenular disease, and has occurred in sympathetic ophthalmia and iridocyclitis. *Premature grayness* of the cilia, sometimes temporary, has also been reported.

[1] Raehlmann believes that trichiasis hairs, or "false cilia," are developed from the epithelial covering of the lid-margin in consequence of marginal blepharitis, the result of granular conjunctivitis.

Entropion, or inversion of the lid, like trichiasis, is most commonly caused in an *organic* form by granular lids, and also follows essential shrinking of the conjunctiva and diphtheritic conjunctivitis. Entropion and trichiasis are often associated.

Two other varieties of entropion are described—*muscular* and *bulbar*. The former is sometimes present at birth from undue development of the orbicularis, and also occurs in a spasmodic type, under the influence of conjunctivitis, keratitis, and foreign bodies; the latter is a falling-in of the lids when the eyeball is shrunken or absent.

Treatment.—The spasmodic varieties will usually subside if the exciting cause can be removed. In temporary entropion the lid may be painted with flexible collodion, which, by its contraction, draws out the inverted border, or, having everted the lid, it may be held in place with a longitudinal strip of plaster which is fastened to the cheek. The organic varieties of the disorder require one or other of the operations described on page 607.

Ectropion, or eversion of the lid with exposure of the conjunctival surface, is either partial or complete. The disorder is divided into the

Fig. 95.—Ectropion of the upper lid, the result of an injury to the brow and subsequent caries of the margin of the orbit (from a patient in the Philadelphia General Hospital).

Fig. 96.—Ectropion of the lower lid, the result of a wound from the tine of a fork (from a patient in the Children's Hospital).

acute or *muscular* and the *chronic* form, or that which results from organic changes.

Acute ectropion usually occurs in children with conjunctivitis and in diseases of the cornea with blepharospasm, when the lids, during examination, become everted and remain so until replaced. One form of partial muscular ectropion is produced by facial palsy.

The common causes of the second, or chronic, form of ectropion are wounds, especially such as are caused by dog-bites, by laceration of the lid by a sharp instrument, by burns and subsequent cicatricial contraction, by chronic inflammatory conditions of the ciliary margin, by ulceration of the lids as in lupus, and by caries of the orbital border and malar bone. The lower lid is more frequently involved than the upper, but ectropion is also seen in the latter position.

Treatment.—This varies with the type and degree of the ectropion. In the spasmodic forms simple replacement of the everted lids suffices;

in slightly marked grades, with some eversion of the lacrimal punctum, the canaliculus should be partly slit, and, if necessary, the nasal duct

Fig. 97.—Ectropion of the lower lid, caused by caries of the malar bone (from a patient in the Philadelphia General Hospital).

Fig. 98.—Ectropion of the upper lid from syphilitic periostitis of the orbit (from a patient in the Philadelphia General Hospital).

should be probed. The organic types of the disorder require a plastic operation for the relief of the deformity (see chapter on Operations).

Certain diseases of the eyelids are comprised in a group of functional disorders of the sebaceous and sweat-glands.

Seborrhea, or that disorder of the sebaceous glands during which their secretion is altered and forms an oily coating on the skin, sometimes accompanied with crusts and epithelial scales, is also seen upon the eyelids. It is usually associated with a similar process in the scalp and eyebrow, and when specially localized upon the ciliary margins creates one of the forms of blepharitis already described.

Treatment.—Proper hygiene, cod-liver oil, iron and arsenic, removal of the accumulated sebum by frequent washings, and the application of sulphur and mercurial ointments comprise the most efficient methods of treatment.

Fig. 99.—Ectropion of the lower lid following lupus. The scar on the cheek is faintly seen (from a patient in the University Hospital).

Milium.—Milia, or small yellowish elevations, consisting of an accumulation of sebum within the distended but closed sebaceous glands,

are common upon the eyelids. They often develop about the age of puberty.

They are caused by improper care of the skin, and may be connected with general constitutional disturbances, dyspepsia, and constipation. They should be opened with a knife or needle and the contents evacuated.

Molluscum contagiosum (**molluscum sebaceum**) is a disease of the sebaceous glands (according to some authors, of the rete mucosum) characterized by the appearance of rounded papules, about the size of a pea, and of a waxy color. The eyelids are a favorite situation.

The disorder occurs chiefly among ill-nourished children, is believed by many to be contagious, and may arise as an epidemic in homes and asylums. According to some observers, the affection is caused by a parasite belonging to the class *coccidia*, and really is a form of *contagious epithelioma*. Muetze's investigations indicate that the "molluscum corpuscles" are the result of a degeneration of the epithelial cells caused by the contagion, the nature of which is uncertain.

Fig. 100.—Dermoid cyst of the eyebrow.

Treatment.—Each molluscum should be incised and its contents forced out.

Ephidrosis (*hyperidrosis*), or an increased flow of sweat, has in rare instances been observed as a local disorder of the sweat-glands of the eyelids. In cases of unilateral sweating of the face the lids necessarily participate. *Retention cysts* of the sweat-glands, in the form of round, sharply circumscribed elevations, have been observed by von Michel.

Chromidrosis (*seborrhœa nigricans*), or the formation of a variously colored secretion from functionally disordered sweat-glands, is sometimes located upon the eyelids. It then receives the name of *palpebral chromidrosis*, and consists of a bluish-black discoloration, usually upon the lower lid, which is somewhat oleaginous and can be wiped away.

It is probably genuine in rare instances; in others it is believed to be either a fraud practised by hysteric subjects or due to the deposit

of dust upon the surface of the skin affected with seborrhea. Young women are usually those affected.

The *treatment* should consist in general invigorating methods calculated to remove anemia, debility, or nervous disturbances. Locally, lead-water and glycerin are recommended.

Sebaceous cysts occur in the eyelids, most frequently in the outer part, and also in the eyebrow. In the latter situation they sometimes are deeply seated, tightly adherent to the periosteum, and may extend some distance into the orbit. *Dermoid cysts* are also found in this region. Their removal by an ordinary dissection is usually unattended with difficulty.

Injuries of the Eyelids.—Incised, lacerated, punctured, and contused wounds, edema, emphysema, and ecchymosis of the lids are the ordinary results of accidents and injuries.

Wounds.—The type of a wound depends largely upon the character of the implement which has inflicted it, and may vary from a simple and superficial incision to a deep cut which penetrates the tissues of the lid and injures the structures of the eyeball. In like manner a laceration may be small and unimportant, or may be so extensive as to tear the eyelid from its attachments. Incised wounds in the line of the direction of the fibers of the orbicularis result in the least visible scar, owing to the absence of gaping. Such injuries are often inflicted by broken glass (a broken spectacle lens, for example) or china, by a knife or other sharp instrument, and by a thin sliver of metal.

Treatment.—Accurate approximation of the edges of the wound should be secured with catgut or silk sutures. Even considerable laceration may heal with very little deformity.

Edema usually occurs as the sequel of a blow, owing to the loose connective tissue of the eyelids, which readily admits of distention (see also page 160).

Treatment.—The application of evaporating lotions, for example, dilute lead-water and laudanum, associated, if the swelling is great, with a pressure bandage, is a measure which will afford relief.

Emphysema of the lids is observed when a fracture of the orbit permits air to escape into the cellular tissue through a communication thus produced with the ethmoidal or frontal sinus. A soft swelling, crackling to the touch, is the result, which increases in degree when the patient blows his nose and forces the air through the fissured bone. The eyelids may participate in the emphysema of the neck and face sometimes seen after tracheotomy or after stab-wounds of the chest.

Ecchymosis of the lids, or a collection of blood in the connective tissue, in its simplest variety constitutes the familiar "black eye," the common result of a blow. A gradual absorption of the effused blood takes place, requiring a week or longer for its completion, but the skin may retain its black-and-blue stain for a greater period of time.

Ecchymosis may be due to fracture of the base of the skull, and may be associated with emphysema if a fracture has involved the frontal or ethmoidal cells.

Treatment.—Emphysema will gradually subside without local treatment; if the swelling is severe, it has been recommended to prick the tissues and allow the air to escape.

Ecchymosis should be treated with frequent applications of cold water, lead-water and laudanum, or diluted white extract of hamamelis. If discoloration remains for a long time, the "eye may be painted." The practice of applying leeches or incising the swollen lid and sucking out the contained blood is to be condemned.

Foreign Bodies in the Eyelids.—Fragments of glass, shot, pieces of iron or steel, portions of wire, particles of stone, and splinters of wood may penetrate the tissue of the eyelids and occasionally remain undetected for long periods of time. They may become encysted or, as the result of infection, give rise to a *lid abscess*. If the foreign body is composed of iron or steel it may be removed by means of a magnet.

Burns of the eyelids are commonly inflicted with hot water, caustics (lye and lime), acids, or are caused by the explosion of powder.

The first agent produces the ordinary vesication, and the treatment should consist in the application of oil, while the pain may be materially relieved by using locally a lotion of carbonate of soda or, better, the moistened powder itself.

Burns caused by the other materials are especially dangerous on account of the almost invariable involvement of the cornea and conjunctiva (see page 239). Immediately after a powder burn all loose powder should be removed, and, if possible, each grain picked out of the skin with a fine needle, or, according to Edward Jackson, destroyed by touching it with a fine electrocautery needle. The application for ordinary burns may then be used. Peroxid of hydrogen has also been employed to remove powder grains, and is most efficient. It may be applied in full strength or in a solution of 3 parts to 1 part of glycerin.

CHAPTER VI

DISEASES OF THE CONJUNCTIVA

Congenital Anomalies of the Conjunctiva.—In addition to dermoid tumors (see page 233) certain thickenings of the conjunctiva of congenital origin have been reported. The latter resemble pterygia and extend between the fissures of the lid (Strawbridge). If necessary, excision could be performed (see also Epitarsus, page 159).

Hyperemia of the conjunctiva (*dry catarrh; hyperæmia palpebraris*) is characterized by an injection of the vessels, chiefly of the palpebral conjunctiva, but rarely affecting the ocular expansion of the membrane. The posterior conjunctival vessels (System I) are involved, but not to the same extent that they are in conjunctivitis. Both an acute and a chronic form exist.

Causes.—The strain of ametropia furnishes a large contingent of these cases, while others arise when the refraction error is insufficiently or improperly corrected. Beginning presbyopia, especially in those persons who are disinclined to use glasses, and hyperemia of the conjunctiva are often associated; it also occurs with incipient cataract and slight opacities of the cornea, as the result of the effort to obtain clear images.

Local irritants, as dust, foreign bodies, misplaced cilia, calcareous concretions, tobacco-smoke, cold winds, etc., are common causes, and the abuse of alcohol originates many cases. The condition may also arise in the eyes of those much exposed to bright light, to great heat—for example, in iron foundries and among workers in *x*-ray rooms. Patients with prominent eyeballs are more liable to hyperemia than those whose eyes are more deeply placed.

Nasal catarrh, lacrimal obstruction, and marginal blepharitis are frequently accompanied by chronic hyperemia of the conjunctiva, which is much aggravated by the establishment of an acute coryza or "hay-fever."

Finally, certain acute hyperemias, which may be recurrent, appear in the form of vasomotor disturbances, and arise under the influence of metabolic disorders, especially gout. Hyperemia of the conjunctiva also occasionally occurs in anemia and chlorosis in place of a pallid membrane, and may be associated with trigeminal neuralgia and migraine. Chronic conjunctival congestion has been attributed to gastro-intestinal auto-intoxication (J. F. Shoemaker).

Symptoms.—Direct inspection reveals the congestion of the vessels, not sufficient to produce the velvety appearance seen in conjunctivitis and unaccompanied by any discharge. Swelling of the conjunctival follicles may be present, especially if the hyperemia is of long standing. There are photophobia, some lacrimation, a hot, stinging sensation

aggravated by the use of the eyes, which readily "water" and grow uncomfortable, especially in artificial light.

Treatment.—This requires the correction of refractive error. Removal of exciting local causes and attention to the anterior and posterior nares are necessary. Patency of the canaliculi and of the lacrimal passages should be secured.

Locally, boric acid, gr. x to f℥j (0.65 gm. to 30 c.c.), or biborate of soda, gr. v (0.324 gm.), camphor water, f℥j (30 c.c.), and distilled water, f℥j (30 c.c.), may be applied. More active astringents, as alum, tannin, and zinc, are sometimes employed, and stimulating drops, as equal parts of tincture of opium and water or boric acid solution, to which a few drops of alcohol have been added, are useful. Nitrate of silver is not advisable; argyrol (10 per cent.) is sometimes useful. Douching the eyes with hot or cold water is a valuable adjuvant. Temporary blanching of the conjunctiva may be secured with adrenalin (1 : 10,000) and preparations of suprarenal extract, but they are not advisable as frequent or constant applications. If there is reason to suspect any general trouble—for example, gout—this must receive attention.

Conjunctivitis.—The conjunctiva is liable to various grades and types of *inflammation* which have certain symptoms in common: (1) Photophobia, not constantly present in all varieties, but commonly seen at some time during the course of the disease; (2) increased and usually altered secretion; (3) a changed appearance in the membrane, varying from a general injection of the blood-vessels and slight velvety opacity to the development of special pathologic products or the formation of false membrane.

The generic term *conjunctivitis* (*ophthalmia* of the older writers) is applicable to this entire group of diseases. Although bacteriologic examinations have given rise to a classification of conjunctivitis which has been recommended in place of the older arrangement, our knowledge is not yet sufficiently exact to make it expedient to banish entirely descriptions based upon clinical appearances. It should be remembered that the normal conjunctiva always contains bacteria, a number of varieties having been isolated. Comparatively few of them should be classified as at all pathogenic (Weeks); but non-pathogenic bacteria may become harmful if the tissues in which they exist are bruised or irritated (Randolph). According to Axenfeld, the *xerosis bacillus* and non-virulent, or only slightly virulent, *Staphylococcus albus* are practically always present in the normal conjunctiva; other organisms occasionally found are *Staphylococcus pyogenes aureus* and *albus*, *pneumococcus*, *Streptococcus pyogenes* (rare), *diplobacillus* and *influenza bacillus* (uncommon), *Bacillus subtilis*, and *sarcinæ*.

Simple Conjunctivitis (*Catarrhal Conjunctivitis or Ophthalmia*).—This is an inflammatory disease of the conjunctiva, characterized by congestion, loss in the transparency of the palpebral conjunctiva, some dread of light and spasm of the lids, and a discharge sufficient only to glue the lids in the morning, or freer and mucopurulent.

Causes.—The etiology is made evident by observing certain varieties:

Associated conjunctivitis is seen with eczema, facial erysipelas, impetigo contagiosa, nasal catarrh, bronchitis, and constitutional disorders like typhoid fever and rheumatism. In *acne rosacea conjunctivitis* minute nodules form at the limbus; moderate irritation supervenes; subsidence takes place, but recurrence is frequent. The condition resembles somewhat phlyctenular conjunctivitis. It arises in adults who are the subject of acne rosacea. General as well as local treatment (dusting with calomel) is required. *Exanthematous conjunctivitis*, which accompanies or follows measles, scarlet fever, and small-pox, may be included in this list. In the conjunctivitis of epidemic meningitis McKee has found the *meningococcus*. This conjunctivitis is an early symptom, and McKee thinks the bacteria may be found in the conjunctival secretion before they can be recovered from the cerebrospinal fluid.

Mechanical conjunctivitis results from exposure to wind, dust, and traumatism (*toxic conjunctivitis*, see page 228).

Symptomatic conjunctivitis may arise from the strain of ametropia, and is analogous to ordinary hyperemia.

Micro-organisms (staphylococci, streptococci, pneumococci) are present in severe types and explain the contagion; neglected hyperemias and the presence of follicular granulations increase the susceptibility to infection, and scrofulous subjects are peculiarly liable to the disease. Occasionally a stubborn conjunctivitis is encountered from the secretion of which pure cultures of staphylococci may be obtained, and which therefore, has been called *Staphylococcus conjunctivitis*. McKee has described a variety of mucopurulent conjunctivitis due to a new pathogenic organism which somewhat resembles the influenza bacillus. A comparatively mild form of conjunctivitis is due to the *Micrococcus catarrhalis*, and is often accompanied by rhinopharyngitis. Conjunctivitis may also be caused by the presence of the *meningococcus*, *Bacterium coli* (Axenfeld), *ozena bacillus*, and *Bacillus subtilis* (Gourfein).

Symptoms.—The secretion is at first watery, and, by running over the edge of the lids, may excoriate the surrounding skin, which shows injection of its superficial veins. In certain individuals the lids, especially along their palpebral margins, are slightly edematous.

The secretion soon becomes mucous or mucopurulent, and, according to the grade of the inflammation, gathers in a slightly frothy material only at the commissural angles, or is more freely secreted.

There are a general hyperemia and loss in the transparency of the tarsal conjunctiva, in which the posterior conjunctival vessels (System I) are concerned, and later of the fornix, caruncle, and semilunar folds.

Although vision is not usually affected, some secretion may be adherent to the cornea and produce the same haziness in sight that would be present on looking through a dirty glass; and artificial lights, which are most uncomfortable at all times, appear fringed with colored borders.

Photophobia may be entirely absent, or exist in marked degree in those varieties which complicate measles, or which are associated with the development of small superficial ulcers on the cornea. All ages of

life are liable to catarrhal conjunctivitis, but the majority of cases are seen in children and young people.

Prognosis and Duration.—The prognosis is perfectly good, and the process will usually subside in a few days. One or both eyes may be affected.

Acute Contagious Conjunctivitis (*Acute Mucopurulent Conjunctivitis; Epidemic Conjunctival Catarrh; "Pink Eye"; Koch-Weeks' Bacillus Conjunctivitis*).—This form of conjunctivitis may be classified as the severe and epidemic type of the variety of conjunctival affection just described. By some writers it is considered as a distinct disease.

Etiology.—The majority of cases are caused by a small bacillus discovered independently by Koch in the acute conjunctivitis of Egypt, and by Dr. John E. Weeks in New York, and studied by Morax and others in Europe. This bacillus resembles that of mouse-septicemia, and measures 1 to 2 μ in length and about 0.25 μ in breadth. It is often associated with a clubbed bacillus (xerosis bacillus). It stains readily with ordinary anilin dyes. Some observers have maintained, but have not demonstrated, that the Koch-Weeks' bacillus and the influenza bacillus are identical, and that acute contagious conjunctivitis is a manifestation of influenza. The disease may occur at any age, except perhaps during the first few days of life, and is widespread over the world. It is commonest in warm and changeable weather (the fall and spring), is markedly contagious, and will pass rapidly from one member of a household to another.

Symptoms.—The period of incubation is about thirty-six hours, and the disease begins with the symptoms of a mild catarrhal conjunctivitis, but usually on the third day develops into a severe form of conjunctivitis, in which the entire conjunctiva is deeply injected and small hemorrhages may be observed (*hemorrhagic catarrhal conjunctivitis*), the swelling of the conjunctival membrane being noticeable in opaque velvety layers, especially in the region of the retrotarsal fold. Sometimes the bulbar conjunctiva is chemotic, sometimes brightly injected. The lids are glued together in the morning, and occasionally they are decidedly swollen and edematous; the eyes are hot and heavy, and feel as though they contained sand. The secretion is at first thick and ropy, and may be gathered into long strings of mucopus. Later, in some cases, the discharge becomes distinctly purulent. The acute stage lasts from four to ten days, and recovery may be expected in about two weeks. Toward the end of the disease, or in what may be known as the subacute stage, the retrotarsal folds are swollen and the papillary body is enlarged. Follicular hypertrophy is at times also observed, and if care is not taken the affection may last for a long time. Both eyes are almost always involved, sometimes simultaneously and sometimes one a day or two in advance of its fellow. Corneal complications are rare, but occasionally occur (Morax, Shumway). Hypopyon has been observed (Morax).

Diagnosis and Prognosis.—The actual diagnosis depends upon microscopic examination and the finding of the specific micro-organism, but the clinical signs are very striking, particularly the character of the

secretion, with its tendency to gather in yellowish masses toward the inner canthus. If the disease is known to be epidemic at the time, or if it is shown to have passed from one member of the family to another, the diagnosis becomes still more certain.

The prognosis is good in the majority of the cases, although relapses and recurrences are common, and one attack does not create immunity. The affection through neglect, however, may prove exceedingly troublesome, and tends to attack all members of a household, a fact which, in asylums and similar institutions, may prove of serious import.

Pneumococcus Conjunctivitis.—This form of conjunctivitis, due to the Fränkel-Weichselbaum diplococcus (pneumococcus), was originally described by Parinaud and Morax, and was supposed by them to be an affection of early childhood; indeed, it is more common in children than in adults, but no age of life is exempt. While it is not as contagious as the Koch-Weeks' bacillus conjunctivitis, its contagious nature is well established, and it may be transferred from one eye to another, from one person to a second, and as Gasparrini, Harold Gifford, Veasey, the author, and a number of other observers have noted, may appear in epidemic form, although not so extensively or frequently as the Koch-Weeks' conjunctivitis. According to Axenfeld, it is more prevalent in northern countries and in cold months of the year than other forms of mucopurulent conjunctivitis. Very rarely it is associated with pneumonia; a coryza may accompany or precede it.

Symptoms.—Usually it begins as an ordinary conjunctivitis, the conjunctiva being reddened and secreting a rather thin mucopurulent discharge, in which small flocculent masses float. The upper lid is edematous and pinkish in color, the lashes lightly matted, and later the discharge becomes thicker and more purulent, and sometimes resembles, in severe cases, that found in purulent conjunctivitis. Small subconjunctival hemorrhages may appear. The clinical manifestations of this form of conjunctivitis are often difficult to distinguish from Koch-Weeks' bacillus conjunctivitis. According to Gasparrini, in pneumococcus conjunctivitis a fine pellicle of fibrin can be wiped from the everted upper tarsus, which is not met with in Weeks' bacillus conjunctivitis. Generally the disease lasts from six to ten days, and the prognosis is favorable; rarely corneal complications have been noted; occasionally iritis develops, due to absorption of the toxin, without implication of the cornea (Axenfeld).

Influenza-bacillus Conjunctivitis.—This form of conjunctivitis, due to the influenza bacillus (Pfeiffer's bacillus), has been especially studied by Zur Nedden, Morax, Jundell, and, in this country, by Arnold Knapp. Clinically, the manifestations of the disease are not severe; it is characterized by a copious, thin discharge, and affects chiefly the conjunctiva of the lids and retrotarsal folds. The majority of cases have occurred in young children and infants, in whom it is more severe than in adults, who are rarely attacked. Although the local disease presents a favorable prognosis, and usually disappears in from ten to fourteen days, it may be associated with rhinotracheitis, dacryocystitis, and

inflammation of the middle ear. Arnold Knapp has described a *pseudomembranous form* of influenza bacillus conjunctivitis of great severity, which may cause perforation of the cornea.

Swimming-bath Conjunctivitis.—This form of conjunctivitis is encountered not infrequently among those who frequent public baths. In many instances the symptoms are analogous to those which occur in catarrhal conjunctivitis, although the discharge is less abundant. Often the bulbar conjunctiva is coarsely injected. In two local epidemics of this disease in two large schools equipped with swimming-pools investigated by the author the microorganism found was in one instance the pneumococcus, and in another an unidentified bacillus. Sometimes the affection resembles acute trachoma, and Paderstein has been able to demonstrate "inclusion bodies."

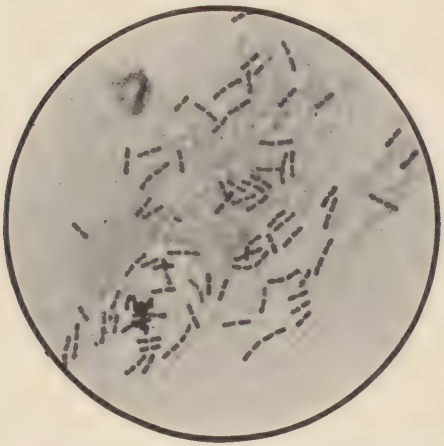


Fig. 101.—The diplobacillus of Morax and Axenfeld (from a preparation by Dr. Harold Gifford).

Diplobacillus Conjunctivitis (*Morax-Axenfeld Bacillus Conjunctivitis, Angular Conjunctivitis, Subacute Conjunctivitis*).—This form of conjunctivitis, due to a diplobacillus 2 to 3 μ in breadth, often occurring in chains, was originally described by Morax and by Axenfeld abroad, and later was studied by Harold Gifford in this country.

Symptoms.—One variety of the disease is insidious in character, and runs a rather tedious course, during which the main symptoms are redness and slight induration of the edges of the lids, particularly of the commissural angles, and congestion of the neighboring conjunctiva. In other words, the signs are those of a *blepharoconjunctivitis*. The abnormal secretion is grayish white and not abundant, and has a tendency to adhere to the reddened lid margins and to gather in small masses at the angles, especially the inner one. This clinical manifestation is so constant that it is well-nigh characteristic, and if there is any doubt, the diagnosis is readily established by finding the bacillus in the secretion. Occasionally these manifestations give place to an *acute form*, or the inflammation may be confined to the conjunctiva without special participation of the lid angle, or the disease may resemble an acute conjunctival catarrh with swelling. According to McKee, it may vary from a mild hyperemia of the conjunctiva to a severe mucopurulent conjunctivitis. Morax-Axenfeld conjunctivitis may attack persons of all ages, but is more common in adults than in young persons. It is widely distributed over the world. It may arise at any season, but is said to be more frequent in summer than during cold weather.

McKee, however, found the greatest number of cases in January. Diplobacillary conjunctivitis may be accompanied by a nasal catarrh, and the diplobacilli are found in the nasal secretion.

Ordinarily the disease lasts from six weeks to six months, but if improperly treated may remain for much longer periods of time. Its duration is readily shortened by proper treatment. It may be associated with follicular and phlyctenular conjunctivitis, and *corneal complications* may arise, either in the form of superficial infiltrations or deep ulcers, from which the bacillus may be cultivated (*diplobacillary keratitis*). Iritis without corneal implication has been observed (Ernest Thomson). Occasionally these ulcers are complicated with hypopyon and iritis (L. Paul). Petit has isolated a bacillus closely allied to the Morax-Axenfeld organism which can produce a conjunctivitis like the one just described, and is also capable of originating hypopyon-keratitis.

The *Bacillus pyocyaneus* sometimes causes a conjunctivitis which yields to zinc sulphate (Brown Pusey).

Treatment of Conjunctivitis.—(*a*) *Simple Conjunctivitis.*—This consists, first, in search for the cause and the alleviation of associated conditions. The patient must be removed from the influence of dust, cold winds, tobacco-smoke, and the like; the under surfaces of the lids should be examined for foreign bodies, and their borders for misplaced cilia. In the earlier stages cold compresses are agreeable and suitable; sometimes frequent bathings with hot water are more acceptable. At first a solution of boric acid, as collyrium or spray—gr. x to f℥j (0.65 gm. to 30 c.c.) is useful. The eyelids and ciliary margins should be frequently washed with water and Castile soap.

As soon as the discharge becomes mucous or mucopurulent and the velvety opacity of the conjunctiva forms, a stronger solution of boric acid, to which a few grains of common salt may be added, is advisable; and the everted lids may be painted with a solution of nitrate of silver, gr. v–x to f℥j (0.324–0.65 gm. to 30 c.c.), and suitably neutralized. In place of nitrate of silver, protargol (5–20 per cent.) and argyrol (10–25 per cent.) are frequently employed. In mild cases one or two applications a day are sufficient, the drug being dropped into the conjunctival sac and allowed to spread freely over the inflamed membrane. Argentamin (2–5 per cent.) and largin (10 per cent.) are also recommended. In severe types, with a considerable discharge, bichlorid of mercury (1 : 8000–10,000) and cyanid of mercury (1 : 2000) are valuable collyria.

Other preparations which have found favor are alum, gr. iv–viij to f℥j (0.26–0.52 gm. to 30 c.c.), sulphate of zinc, gr. ij to f℥j (0.13 gm. to 30 c.c.), which may be suitably combined with boric acid, biborate of sodium, gr. iv–viij to f℥j (0.26–0.52 gm. to 30 c.c.), peroxid of hydrogen, Panas' fluid, creolin (1 per cent.), and other antiseptic collyria. Should the thickening of the retrotarsal folds prove stubborn, after the acute symptoms have subsided, these may be touched with an alum crystal or a solution of tannin and glycerin.

Atropin is not usually necessary unless a corneal ulcer complicates the affection.

The eyes may be protected with smoked glasses, but in no circumstances should they be bandaged or be covered with poultices of tea-leaves (which of themselves may produce conjunctivitis—"tea-leaf conjunctivitis"), bread and milk, scraped potatoes, and the like. It should be remembered that meddlesome domestic medication of this sort may change a simple conjunctivitis into a serious and purulent inflammation.

At the outset a laxative, followed by full doses of quinin, is indicated; any associated disease of which the conjunctivitis may be a symptom—*e. g.*, rhinitis—requires the usual treatment. Proper hygiene, fresh air, strict cleanliness, and protection from contaminated towels, etc., are evident indications.

(*b*) *Acute Contagious Conjunctivitis.*—The treatment does not differ from that which has already been described in connection with simple conjunctivitis. Weeks recommends bichlorid of mercury (1: 10,000) as a collyrium. Usually argyrol and protargol are prescribed, and they may act efficiently, but it is a mistake to permit patients themselves to use these preparations for long periods of time. As Risley has aptly said, they may originate a new set of conjunctival catarrhs, and give rise to a troublesome, tumid, hyperemic condition of the conjunctiva; also they may stain the membrane. Applications of nitrate of silver, in the usual manner, to the everted lids are useful. Iced compresses afford relief during the height of the affection. In place of the ordinary collyria, chlorid of zinc, 1 grain (0.065 gm.) to the ounce (30 c.c.), is highly commended; sulphate of zinc, gr. ij to f ʒj (0.13 gm. to 30 c.c.), is also valuable.

(*c*) *Pneumococcus Conjunctivitis.*—In this affection the treatment already described in the preceding paragraphs is efficient. Sulphate of zinc drops are especially useful.

(*d*) *Influenza-bacillus Conjunctivitis.*—The usual collyria and antiseptics are advisable. Zur Nedden recommends nitrate of silver (1.5–2 per cent.), oxycyanid of mercury (1 : 1500), and iced compresses.

(*e*) *Diplobacillus Conjunctivitis.*—In this disease the preparations of zinc are practically specifics. They may be employed in the form of the sulphate (0.5–2.5 per cent.) or the chlorid (0.2 per cent.). Nitrate of silver, argyrol, and protargol, in contradistinction to their valuable action in other varieties of conjunctivitis, are of no use. Todd recommends sozoiodolate of zinc (1–2 per cent.) and ichthyol ointment (10 per cent.).

Catarrhal Epidemic Conjunctivitis (*Catarrh with Swelling; Epidemic Catarrh*).—Certain systematic writers, notably Saemisch and Schmidt-Rimpler, although they consider this disease to be a form of acute conjunctival catarrh, give it a separate description on account of certain distinguishing features, namely, swelling not only of the lid margin, but of the entire lid itself, which seems to be increased in volume and is reddened, together with tumefaction, infiltration, and marked hyperemia of the retrotarsal folds. The secretion is considerable in quantity, mucopurulent in character, and often mixed with small fibrinous

masses. Both eyes are usually affected, and persons of any age may be attacked. It has appeared with especial frequency among scrofulous children, especially if they are also the subjects of exanthematous disease of the face. The secretion should be considered as distinctly contagious, and the disease has appeared in the form of small epidemics. Sometimes it has been the sequel of influenza. In the secretion, pneumococci, staphylococci, and streptococci have been found. The treatment should include thorough irrigation of the conjunctival sac with bichlorid of mercury (1 : 8000), cyanid of mercury (1 : 1500), or a saturated solution of boric acid. The swollen and inflamed retrotarsal folds may be treated with applications of 1 to 2 per cent. solutions of nitrate of silver in the usual manner, or in their place protargol and argyrol may be employed.

Exanthematous Conjunctivitis.—This form of conjunctivitis has been briefly referred to in connection with catarrhs of the conjunctiva, and is particularly noteworthy as part of the manifestations of measles, scarlet fever, and small-pox. The disease may not only accompany these exanthems, but often arises prior to their eruption. Small-pox pustules sometimes develop on the conjunctiva. Occasionally the conjunctivitis of measles assumes a very severe type, so severe that it may resemble a blennorrhea, and Fuchs has noted under these conditions a suppurative inflammation of the Meibomian glands. The investigations of Schottelius indicated that staphylococci and streptococci are active in the conjunctivitis of measles, streptococci being especially common in fatal cases.

Among the chronic exanthemata of the conjunctiva, *acne rosacea* is important (see also page 189), and in this disease minute nodules may form with marked irritation near the limbus. The conjunctiva may also be implicated in pityriasis, psoriasis, and herpes iris. Its involvement in syphilis, pemphigus, lupus, and lepra is elsewhere described.

Treatment.—The treatment of exanthematous conjunctivitis does not differ from that of the catarrhal form of the affection and its varieties. Fuchs recommends the local application of calomel as especially valuable in acne rosacea of the conjunctiva.

A very rare form of conjunctivitis or conjunctival disease is "squirrel plague conjunctivitis," described by Derrick Vail and Robert Sattler, due to an infection with the *Bacillus tularense*, identified by McCoy and Chapin as the germ of squirrel plague. In Vail's patient the symptoms were: intense chemosis, mucoid secretion, enlarged preauricular glands, and deep yellow necrotic ulcers in the palpebral conjunctiva.

Purulent conjunctivitis (*acute blennorrhea of the conjunctiva*) occurs in three specific forms: in the newborn (*ophthalmia neonatorum*), in young girls (occasionally boys), and in adults (*gonorrheal conjunctivitis* or *ophthalmia*).

Conjunctivitis Neonatorum (*Ophthalmia Neonatorum*).—This is an inflammation of the conjunctiva, characterized, in its usual form, by

great swelling of the lids, serous infiltration of the bulbar conjunctiva, and the free secretion of contagious pus.

Causes.—The affection is most frequently caused by the introduction into the eye of the infecting material from some portion of the genito-urinary tract of the mother at the time of or shortly after birth. The majority of cases (60–70 per cent.) are associated with a special micro-organism—the *gonococcus of Neisser*. Exceptionally, inoculation occurs *in utero*, owing to the penetrating power of the gonococcus or to infection after rupture of the membranes (*antepartum conjunctivitis*).

Many observers have demonstrated that ophthalmia neonatorum is not always gonorrheal in origin, but may be produced by various kinds of micro-organisms—pneumococcus, streptococcus, diplobacillus, Bacterium coli, and Staphylococcus albus, aureus, and citreus. It is possible that some forms of conjunctivitis neonatorum are really types of influenza-bacillus conjunctivitis. According to Stephenson, next to the gonococcus the pneumococcus is the commonest organism found with ophthalmia neonatorum. *Inclusion-blennorrhea of the newborn* is referred to elsewhere (see page 203).

Inasmuch as the gonococcus is not invariably present, two forms of the disease have been distinguished—a severe type, caused by the gonococcus, with a tendency to increase in severity and invade the cornea; and a milder type, non-specific, with a tendency to recover. Hence, a virulent vaginal discharge is not necessary to produce this condition, except in intense degree, and it probably may arise from the contamination of any mucopurulent discharge during birth, and from injudicious intravaginal antisepsis with strong solutions of mercuric chlorid. Careless bathing of the child after birth and the use of soiled towels and sponges are fruitful sources of infection. Contact with the lochial discharge may originate the disorder, although inoculation with healthy lochia has failed to produce the disease.

The exact time of inoculation has not been determined. Infection is more likely to occur in face presentations and during retarded labors. Boys are attacked more frequently than girls. The disease is said to be more common during summer months in cold climates; in hot countries, during the spring and autumn.

Symptoms.—Conjunctivitis neonatorum usually begins on the third day after birth, but may set in as early as from twelve to forty-eight hours after inoculation, or, when it is the result of a secondary infection from soiled fingers, sponges, or clothes, be delayed to a much later date. Late gonococcic infections have been explained by Crede-Hörder on the assumption that the Neisser organisms do not find their way directly into the conjunctival sac during birth, but may be hidden, for example, in Meibomian glands. Non-gonorrheal cases do not usually arise until after the fifth to the seventh day. Almost always both eyes suffer, the one being earlier and frequently more decidedly affected than its fellow.

Four stages of the disease are common, but as these vary in different

cases, and more or less rapidly shade one into the other, no very sharp lines need be drawn.

A slight redness of the conjunctiva, with a trifling discharge in the corner of the eye, is rapidly succeeded by great, cushion-like swelling of the lids, with intense chemosis and congestion of the conjunctiva, accompanied by severe pain and discharge. The surface of the swollen lid is hot, dusky red, and tense; the upper lid overhangs the lower, and at first can be everted only with difficulty. The discharge, which in the beginning is slightly turbid, soon changes to a yellow or greenish-yellow pus, and is secreted in great quantities.

If the lids are everted during the first day or two of the disease, the conjunctiva will be found to be swollen, red, and velvety, and that upon the eyeball intensely injected; upon the surface easily detached

Fig. 102.—Conjunctivitis neonatorum (from a patient in the Philadelphia General Hospital).

flakes of lymph are found; later, the conjunctiva becomes rough and of a dark-red color, spots of ecchymosis appear, or it is succulent and bleeds easily. Marked chemosis and infiltration of the ocular conjunctiva succeed, forming a hard rim; at the bottom of the crater-like pit thus produced the cornea may be seen. The thick, cream-like discharge increases, and either flows out from beneath the overhanging upper lid on to the cheek or is packed up in the conjunctival culdesac (Fig. 102). Sometimes false membrane forms and covers the tarsal conjunctiva; indeed, the appearances may be exactly like those of a *membranous conjunctivitis*.

The lids now may lose much of their tense character, and can be more easily everted; the conjunctiva is puckered into folds and papilla-like elevations, and the discharge contains an admixture of blood and serum. Gradually the disease declines, and in from six to eight weeks

the discharge ceases. The relaxed palpebral conjunctiva is thick and granular, looking like the granulation tissue which surrounds wounds. The ocular conjunctiva is also thickened, and positive cicatricial changes may remain.

The chief risk is destruction of the vitality of the cornea, the danger of which is materially increased if this membrane becomes lusterless, dull, and hazy within the first day or two of the disease, and the gonococcus is freely present in the discharge. Frequently small oval ulcers form near the limbus, either transparent or surrounded by an area of cloudy infiltration, which rapidly increase in size; or larger areas of ulceration develop in a more central situation. In many mild cases the cornea escapes without harm. The changes which take place in the cornea are due in part to strangulation of its nutrient vessels by the swollen tissue, but largely to direct infection by the discharge. Corneal lesions do not usually occur in eyes if the discharge is free from gonococci.

After the formation of a corneal ulcer, either its healing and regeneration of the corneal tissue takes place or else perforation occurs.

The result of perforation will depend upon the amount and character of the destruction of the corneal tissue. If the ulcer is central and perforates, the aqueous humor escapes, the lens is pressed forward against the posterior surface of the cornea, and the opening becomes closed with lymph. Restoration of the anterior chamber follows, and the lens returns to its proper position, carrying with it upon the anterior capsule a little mass of lymph. Thus the formation of a *pyramidal cataract* results (see page 404). Fuchs maintains that in the formation of opacities following corneal suppuration the lens epithelium is rarefied by destruction of the cells; later there is proliferation of the remaining cells and *capsular cataract* results.

Perforation of an ulcer peripherally situated, especially below, is followed by adhesion of the iris to the opening. The aqueous escapes, and, as the iris and the lens fall forward, the former becomes entangled in the perforation and is fixed by inflammatory exudation. The adhesion is either on the posterior surface or in the cicatrix, and the resulting dense white scar receives the name *adherent leukoma*.

If the region of the scar is bulged forward because it is unable to resist the intra-ocular tension, *anterior staphyloma* results. Extensive sloughing of the corneal tissue, with total prolapse of the iris, matting together of the parts by exudation, and protrusion of the cicatrix constitute a *total anterior staphyloma*.

Finally, perforation may be followed by inflammatory involvement of the ciliary body and choroid, and the rapid destruction of the eye through *panophthalmitis*, or a slower shrinking of the tissues, with *atrophy* of the *bulb*. Dense opacity occasionally appears in the cornea during convalescence, and may go on to ulceration or clear up perfectly. It may arise with great suddenness, and, should it occur in the lower half of the cornea, a deep indentation, owing to the pressure of the margin of the lid, is likely to occur.

The appearance of the conjunctiva differs materially in different

cases. Its surface may be covered over not merely with easily detached flakes of lymph, but with a gray, false membrane. More rarely a deep infiltration develops, like that seen in diphtheritic conjunctivitis.

Restlessness, fever, and other constitutional disturbances are sometimes present, and synovitis of the knee and wrists may arise, of the same character as arthritis occurring in adults during gonorrhea. Rhinitis, infection of the lacrimal gland, meningitis, endocarditis, and general septicemia have been reported as complications of ophthalmia neonatorum. In rare instances pneumococcus conjunctivitis neonatorum may be associated in the second week with inflammation of the knee-joint (Stephenson).

Conjunctivitis neonatorum does not always follow this course, because the term is made to include affections of the conjunctiva in the newborn other than the types just described—mild catarrhal conjunctivitis, hyperemias, and that variety which, according to Noyes, presents the character of a granular, rather than of a purulent, conjunctivitis, and which may continue for weeks without danger of corneal complication. Occasionally a gonococcal conjunctivitis pursues the course of a simple conjunctival catarrh (Groenouw). Furthermore, as Saemisch has said, purulent conjunctivitis may develop in newborn children not due to the gonococcus (see also page 196), but caused either by other virulent bacteria or by non-bacterial agents. Necessarily the manifestations are less violent than those of gonococcal origin, and for these varieties the name *acute blennorrhagic conjunctivitis neonatorum* has been suggested.

Some hyperemia of the conjunctiva, with a little yellowish discharge in the corners of the eye and slight swelling of the lower lid, is common in babies for a few days after birth.

Diagnosis.—The onset and character of the typical disease, its symptoms and course, render a mistake in regard to its nature practically impossible. Close attention should be given to what at first appears to be a trivial inflammation in the eyes of a newborn child, because a virulent and destructive inflammation may follow with great rapidity. Bacteriologic examination of the secretion is essential, and the findings will determine the true character of the disease.

Prognosis.—This is always grave in gonorrheal cases, but with competent medical attendance, *if the eye is seen while the cornea is still clear*, except in diphtheritic types, in those with inherent malignancy (Randall), or where depreciation of nutrition or intercurrent illness diminish the resisting power of the child, the majority of cases should be brought to a successful termination. Hence the attendants of newborn children should be compelled to seek medical advice as soon as conjunctival trouble appears, for delayed or improper treatment means sloughing of the cornea, when no form of medication can do more than relieve the violence of the inflammation, which, after it subsides, leaves the child with sight hopelessly marred, perhaps destroyed. The prognosis of the mild types is favorable.

Prophylaxis.—The present high standard of scientific midwifery

includes such cautious antisepsis during labor that the risk of contamination is distinctly less than in former times, but still some preventive method should be employed.

The eyes of those children who have passed through a birth-canal known to be infected, or from which the suspicion of infection cannot positively be eliminated prior to birth, may be treated according to the method of Credé, which is as follows: As soon as the head is born the lids are carefully cleansed, parted, and two drops of a 2 per cent. solution of nitrate of silver are instilled into each conjunctival sac. Small cold compresses are then laid upon the lids and renewed at suitable intervals. Occasionally severe reaction follows—conjunctival hyperemia or catarrh (the so-called "silver catarrh"), and even hemorrhage from the conjunctiva and corneal haze. Hence it is not necessary to employ this method in all cases, and even in those in which it is indicated a 1 per cent. solution of nitrate of silver is sufficient. Wherever infection, or the suspicion of infection, can be excluded, milder measures —for example, washing the eyes and flushing them with a saturated boric acid solution—are sufficient. In place of a 2 per cent. solution of nitrate of silver a 1 : 500 solution may be used. Other materials recommended are aqua chlorini (Schmidt-Rimpler), sophol (5 per cent.) (von Herff, R. M. Williams), bichlorid of mercury (1 : 5000), carbolic acid (1 per cent.), and the newer silver salts, especially argyrol (25 per cent.) and protargol (10 per cent.). The last-named remedies are not to be trusted in the management of eyes which have been exposed to gonorrheal infection. The value of Credé's method is so firmly established that it should not be neglected if the birth-canal is known to be infected with gonorrhea, or if the suspicion of infection cannot be excluded. In its place a 5 per cent. solution of sophol has been highly recommended. Axenfeld advises its use "instead of the formerly employed Credé's silver drops." With this drug the author has had no experience. The hands of the mother, nurse, and child should be searched for sources of infection, and, if gonorrhea is known to exist in the mother, the child should be isolated. Conjunctivitis neonatorum should be listed as a reportable disease, and laws should be enacted to this effect, and these laws should be rigidly enforced, and it should be possible to provide the affected children with expert attention at their homes or, even better, with hospital accommodations. The distribution by health boards of circulars of advice to midwives and mothers and of tubes containing the chosen prophylactic, to those who are qualified to use it (preferably a 1 per cent. solution of nitrate of silver), should be required (F. Park Lewis, J. Clifton Edgar, and F. F. Wesbrook). As ophthalmia neonatorum is the cause of fully 8 per cent. of blindness in this country, all efforts to check the ravages of this disease cannot too strongly be emphasized.

Treatment.—If the type is mild, the applications described under simple conjunctivitis are indicated; if severe, three conditions demand attention: the inflammatory swelling of the lids, the state of the conjunctiva, and the corneal complications.

1. During the earlier states, when the lids are tense and the secretion lacking in its later creamy character, in addition to absolute cleanliness, local application of cold is a useful agent in a certain number of cases.

This should be applied in the following manner: Upon a block of ice square compresses of gauze are laid, which, in turn, are placed upon the swollen lids, and as frequently changed as may be needful to keep up a uniform cold impression. This is far preferable to the use of small bladders containing crushed ice; indeed, the use of ice is not advisable. The length of time occupied with these cold applications must vary according to the severity of the case. Sometimes they may be used almost continuously, and sometimes every three or four hours for twenty minutes at a time. Standish and other surgeons deny the value of the use of cold in this manner, believing that it adds to the danger of corneal complications. This is entirely contrary to the author's experience.

It must be emphasized, however, that it requires a good deal of experience to know when to use and when not to use cold, and not all cases are suited to its application. Hot fomentations have been advised, especially where corneal complications exist, or the surface of the conjunctiva is covered with a gray film. These are applied with squares of antiseptic gauze wrung out in carbolized water of a temperature of 120° F., and frequently changed. The author, basing his opinion on a large experience in the Philadelphia General Hospital, doubts their efficiency.

2. Constant removal of the discharge must be practised.

The lids are to be gently separated, the tenacious secretion wiped away with bits of moistened lint or absorbent cotton, and the conjunctival sac gently but freely irrigated with an antiseptic fluid. For this purpose a saturated solution of boric acid (which is feebly antiseptic, but very cleansing and slightly astringent) or one of corrosive sublimate, 1 grain to 1 pint—0.065 gm. to 480 c.c. (strong solutions should not be used, because they may injure the corneal epithelium and cause ulceration), may be employed. Special and ingenious forms of lid irrigators have been devised, but are unnecessary and often harmful. The cleansing process must be repeated at least every hour, day and night, and, if necessary, much more frequently; but all manipulations must be most gentle and all caution not to injure the delicate structures of the eye must be maintained.

3. The application of one of the salts of silver.

Until comparatively recent times nitrate of silver was almost universally employed, a drug combining the properties of an astringent and a superficial caustic. Its germicidal power, however, is not of much avail, as this is quickly impaired by contact with the tissue proteins. Once a day the palpebral conjunctiva and retrotarsal folds should be brushed over with a solution, 10 grains (0.65 gm.) to the ounce (30 c.c.) their surfaces first having been carefully freed from any adherent discharge, and afterward all excess of the drug washed away with a solution of common salt, and this washing continued until a clean red surface is secured, when the lids may be returned to their proper posi-

tion, their margins greased with vaselin, and some of the lubricant introduced within the conjunctival culdesac. Great care must be exercised that the corneal epithelium shall not be injured. Ulceration of the cornea does not alter the treatment described. As long as the discharge is abundant the use of the caustic is indicated, and it may be employed from the very beginning of the disease unless the conjunctiva is covered with a false membrane, which would prevent its access to the conjunctival folds.

Within the last few years protargol and argyrol have largely replaced nitrate of silver in the treatment of ophthalmia neonatorum in the practice of many surgeons, and for noteworthy reports on their efficiency we are largely indebted to Myles Standish. His routine treatment is as follows: The edges of the lids are washed with a solution of boric acid once in half an hour, and they are anointed with vaselin to prevent them from sticking together. A solution of protargol or argyrol is instilled freely between the lids at intervals of from every hour to once in four hours. This fluid sinks to the bottom of the culdesac and floats to the surface the pus and mucus, which can readily be removed with a very slight amount of manipulation. Protargol has been used in strengths varying from 10 to 40 per cent., the 10 per cent. solution yielding the best results. Of argyrol, a 25 per cent. solution proved to be satisfactory. Henry D. Bruns recommends a 10 per cent. solution of argyrol to be used freely every half-hour until pus secretion is checked. Then he applies in the usual manner nitrate of silver (0.2–1.0 per cent.) once a day. Many other surgeons, both here and abroad, employ these drugs in a similar manner. According to Stephenson, in gonorrheal ophthalmia neonatorum a 25 per cent. solution of argyrol painted once or twice a day over the conjunctiva, exposed for that purpose by eversion of the lids and carefully dried from adherent discharge, with the frequent use by instillation of the 25 per cent. or of a weaker solution, represents a method of treatment more promptly efficacious than any other with which he is acquainted.

The author's experience is in accord with the good results ascribed to the methods of using argyrol just described, only in so far as the comparatively mild cases of ophthalmia neonatorum are concerned in which this drug achieves good results, not because its action is more efficient in controlling the disease than that of nitrate of silver, but because it is more easily applied, is less irritating, and has a marked detergent effect, driving the secretion before it. In a number of cases the argyrol treatment will not be sufficient, and nitrate of silver must be used, especially, as Bruns recommends, in addition to or after the use of argyrol. The author is entirely unconvinced that argyrol should replace nitrate of silver, although he practically employs it as part of the treatment in suitable cases.[1] Protargol possesses no advantages over nitrate of silver.

[1] The author is well aware that many surgeons deprecate the use of nitrate of silver in these circumstances. George Derby, for example, thinks the reaction in gonorrheal conjunctivitis is too great to permit its use and that its bactericidal power cannot assail the deep-seated gonococci. He prefers mild remedies, *e. g.*, argyrol. E. B. Heckel reports excellent results from constant irrigation alone with iced physiologic salt solution.

At the first appearance of corneal haze one drop of a 0.5 per cent. solution of atropin is to be dropped into the eye two or three times daily. During corneal complications Darier recommends a collyrium containing dionin, pilocarpin, and cyanid of mercury.

Persistent swelling of the conjunctiva is sometimes treated by scarification. Division of the outer commissure to relieve pressure is not suited to young infants, although it may be indicated in adults.

If one eye alone is affected, suitable protection for the sound eye should be provided. This may be accomplished by antiseptic bandaging of the uninflamed organ (Buller's shield is difficult of application in infants).

The attendants must be impressed with the fact that upon their faithful carrying out of directions, and upon their unremitting care, much, if not all, of the hope of bringing the case to a successful termination depends. The attendants must further be impressed with the contagious nature of the pus; all bits of rag and pledgets of lint used in the treatment must be destroyed, and after each treatment the hands of those engaged must be thoroughly washed and disinfected with a solution of bichlorid of mercury.

Many other remedies have been used in the treatment of conjunctivitis neonatorum; for example, those mentioned on page 193: carbolic acid (0.5 to 2 per cent.), iodoform and iodoform ointment (4 per cent.), aqua chlorini, cyanid of mercury (1 : 1500), permanganate of potassium (1 : 5000), used in copious irrigations, formaldehyd (1 : 5000), and argentamin (2 per cent.). Darier suggests a 3 per cent. solution of ichthargan if protargol loses its effect. Blenolenicet salve has been recommended (Adams, Scheuermann), and sophol in 5 per cent. solution (Von Herff). Antigonococcus serum has been tried and good results have been reported. These remedies do not seem to possess virtues which should make them replace those which have been more fully described.

Inclusion-blennorrhea of the Newborn.—In certain varieties of conjunctivitis neonatorum, usually non-gonorrheal and for the most part benign in nature, the Giemsa stain will reveal in the secretion "epithelial inclusions," that is, clusters of small granules, colored violet or dark blue, which cap the nucleus like a cowl. According to Axenfeld, this "inclusion-blennorrhea" is frequently unilateral, the retrotarsal fold is tumid, and if the disease lingers long a granular condition of the conjunctiva supervenes. After its subsidence sometimes delicate cicatricial tissue remains. Corneal complications are not in evidence. Whether these "inclusions" are the cause of this disease is not known (see also page 217).

Purulent Conjunctivitis in Young Girls.—Occasionally young girls are the subjects of vaginitis, which in severe forms is associated with a purulent discharge, and in hospitals and asylums has occasionally assumed the form of an epidemic among the inmates. In a certain percentage of these cases gonococci are present in the discharge, and the disease may be conveyed to the eye by the fingers, or gain entrance into the conjunctival sac from discharge adherent to bed-linen, sponges, etc.

There results a purulent conjunctivitis, with symptoms closely resembling those of ophthalmia neonatorum, although usually the manifestations are less violent, and the corneal complications less likely to occur than in the gonorrheal conjunctivitis of adults. To this disease the name *ophthalmoblennorrhea*, or *gonoblennorrhea, of young girls* has been given. The treatment should in all respects conform to that which has been described in connection with ophthalmia neonatorum. If properly treated, the prognosis is good.

Gonorrheal conjunctivitis (*purulent ophthalmia; acute blennorrhea in adults*) usually can be traced to its source of contagion from an acute gonorrhea or a gleet, by contact with soiled fingers or linen, or from an eye affected with this form of conjunctivitis.

The same micro-organism described in connection with gonorrheal ophthalmia neonatorum is active in gonorrheal conjunctivitis, the diplococci being found within the cells; later they penetrate the epithelium and enter the lymph-spaces in the subconjunctival tissue.

Symptoms.—The first symptoms appear from twelve to forty-eight hours after inoculation, and resemble those already recited in connection with the same disease occurring in the newborn (see page 196).

The vitality of the cornea is in constant danger, and involvement of this membrane may arise during the height of the attack or later, and when convalescence apparently is established. This consists in ulcers, small and large, either central or peripheral; in the latter position they often exist as grooved rings or small clean-cut lesions without infiltration, hidden by the swelling of the surrounding conjunctiva, and very prone to perforate. A more or less dense opacity may follow ulceration or arise independently of this condition.

If perforation occurs, all the phenomena described on page 198 will ensue, and even without perforation, iritis, cyclitis, and disease of the deeper structures of the eye may develop and defeat the possibility of obtaining good vision.

Gonorrheal conjunctivitis reaches its climax in about ten days and then gradually subsides in from one to two months; or it may pass into a chronic type and be one of the forms of *chronic blennorrhea*, in which there is general redness of the palpebral conjunctiva, with hypertrophy of its superficial layers and some thickening of the papillæ.

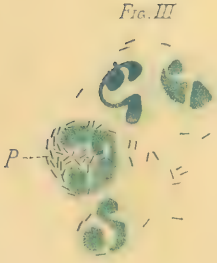
Diagnosis.—This is readily made from the history of the case, and, above all, by an examination of the secretion for the gonococci.

Prognosis.—The prognosis is *always* grave, even more so than in conjunctivitis neonatorum. Fully developed gonorrheal conjunctivitis rarely recovers without some corneal involvement, and only too often the eye is hopelessly marred. Arthritis, endocarditis, and septicemia may arise as complications (see also page 199).

Treatment.—This includes the same principles and practice described in connection with ophthalmia neonatorum (see page 200), but requires certain modifications suggested by the adult age of the majority of the patients.

If the swelling of the lids is so great that their pressure threatens

PLATE II.



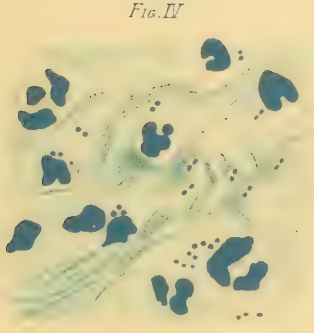


FIG. I.—Discharge from right eye in a case of purulent conjunctivitis; gonococci numerous in cells (Stephenson).

FIG. II.—Bacillus of Weeks in pure culture (from a photograph) (Weeks).

FIG. III.—Conjunctival secretion from acute contagious conjunctivitis; polynuclear leukocytes with the bacillus of Weeks; *P*, Phagocyte containing bacillus of Weeks; immers. $\frac{1}{12}$, oc. iii (Morax).

FIG. IV.—Secretion from a case of conjunctivitis, showing pneumococci; immers. $\frac{1}{12}$, oc. iii (Morax).

to destroy the cornea, the outer canthus may be divided (canthotomy). This acts in a twofold manner, by relieving pressure and by depleting the engorgement through the loss of blood occasioned by the incision, which should be made with a scalpel, cutting the tissues from without down to the bone as far as the margin of the orbit, but leaving the conjunctiva uninjured. Repeated incisions of the hard rim of chemotic conjunctiva which surrounds the cornea will also relieve pressure, and in some circumstances is a most useful procedure. In desperate cases some operators (Critchett, Fuchs) have not hesitated to split the lid vertically and stitch the divided portions to the brow, restoring them by a plastic operation after the disease has subsided. Cold may be applied with compresses in the manner already described, or continuously with Leiter's tubes. Certain experienced surgeons, as has already been mentioned in connection with ophthalmia neonatorum, doubt the value of cold applications. The author believes that in gonorrheal conjunctivitis in adults, during the early stage, cold is not only most agreeable to the patient, relieving pain and irritation, but of distinct value in checking the inflammatory process and the movement of the bacteria. Not all cases are suited to cold, and it is not always right to use the cold continuously; but, as is well maintained by Weeks, it may be used for periods of twenty minutes to half an hour every three or four hours, thus obtaining the therapeutic value of the cold and avoiding the danger of depressing the nutrition of the cornea.

Local applications include the antiseptic lotions previously given (see page 202), in addition to which may be mentioned a drug which the author can highly recommend, namely, permanganate of potassium, 1 : 2000–5000, used copiously, a pint at a time, in continuous irrigation after the manner of Kalt. These irrigations should be performed three or four times a day, according to the severity of the case and the quantity of the discharge. Nitrate of silver should be employed in the manner described on page 201. In place of this drug argyrol and protargol have been much employed. According to Standish, protargol seems to act somewhat better in the gonorrheal conjunctivitis of adults than argyrol. If either of these drugs is used it should be applied in the manner already described (see page 202). In the opinion of the author, argyrol, if used at all, should be employed only as an adjuvant in the treatment of gonorrheal conjunctivitis, and not to the exclusion of nitrate of silver, which, he is satisfied, will always hold a high position in the therapy of this disease in adults. With subconjunctival injections in the treatment of serious cases of gonorrheal ophthalmia, as, for example, they have been recommended by Hirsch, who uses a solution of oxycyanid of mercury, 1 : 5000, to which acoin is added, the author has had no experience. Neither has he faith in the value of peroxid of hydrogen diluted to 3 per cent., which has been recommended in the treatment of this disease, nor has he had an opportunity of testing the value of blenolenicet ointment. The treatment of this disease with antigonococcus serum does not appear to have been satisfactory.

On the appearance of any of the types of *corneal ulceration* atropin drops should be instilled with sufficient frequency to maintain mydriasis and subdue ciliary hyperemia; indeed, it is a wise precaution to employ this mydriatic from the beginning of the disease. Eserin has also been recommended, or the combined action of eserin and atropin, obtained by using the former drug during the daytime and the latter at night. In the majority of instances atropin will secure the best results. Iodoform freely dusted upon the ulcer is of service, and other measures mentioned on page 253 must be considered. Dionin is recommended by Darier.

If an ulcer threatens to perforate, paracentesis (see page 619) through its floor will diminish the intra-ocular pressure, and may prevent or lessen the extent of prolapse of the iris. A substitute for this operation is the use of the actual cautery or an application of pure carbolic acid, and should be preferred. If perforation has taken place, excision of the prolapsed iris, sometimes recommended, is not without danger, as this procedure may open a way for the entrance of infecting material to the deeper structures of the eye. The final outcome of the case will depend upon the extent of corneal involvement and the ultimate treatment of the remaining leukoma, staphyloma, or shrunken ball will require, according to circumstances, iridectomy, abscission, evisceration, or enucleation.

Often the patients are debilitated, and supporting treatment is indicated, namely, quinin, iron, strychnin, and milk-punch. If there is constipation, calomel and saline laxatives should be administered. The pain, which is often severe, may be allayed with morphin or opium. In place of morphin, codein may be employed. It is a mistake, in the serious forms of this disease, to depend alone upon local measures.

The treatment of a *chronic conjunctivitis*, the sequel of an acute attack, depends upon the degree of thickening in the mucous membrane, but is usually best managed by careful exposure of the thickened conjunctiva and applications of nitrate of silver, tannin and glycerin, and the occasional use of the alum or sulphate of copper stick. A collyrium of boric acid, bichlorid of mercury, or sulphate of zinc may be used.

Prophylaxis.—Patients suffering from gonorrhea should be warned not only of the great danger of infecting their own eyes, but the eyes of those around them. Inasmuch as a very minute quantity of urethral discharge, and even where this is the product of a chronic disease—gleet, for example—may produce acute conjunctivitis, these precautions become the more necessary.

As usually one eye alone is affected, it is a matter of great importance to secure the other eye from contact with the secretions. This may be done by sealing it with an antiseptic bandage, the edges of which are made tight by fastening along them strips of gauze painted with flexible collodion, or by the application of Buller's shield. The latter consists of a watch-glass fitted in a square piece of rubber adhesive plaster, which is carefully applied to the brow, temple, lower margin of the orbit and

nose, and should be secured with additional strips to prevent the entrance of discharge.[1] The inner margin should be sealed with collodion, as a contamination is most likely to occur at this point, and inefficient application increases, rather than diminishes, the danger.

All the precautions which have been stated in regard to the care of conjunctivitis neonatorum apply with equal and even greater force to the present disease. In a number of instances the surgeon in attendance has been contaminated.

Metastatic Gonorrheal Conjunctivitis[2] (*Gonorrheal Epibulbar Conjunctivitis*—Heerfordt).—This is an inflammation of the mucous membrane of the eye due to the gonococcus which is carried from the urethra to the conjunctiva through the medium of the circulation; or it may be caused by the gonotoxin. According to W. Gordon M. Byers, whose definition has just been quoted, it may occur as the initial symptom of a generalized infection, appear simultaneously with the other manifestations—for example, arthritis—follow the outbreak of inflammatory symptoms elsewhere located, or be the only expression of a systemic gonorrhea.

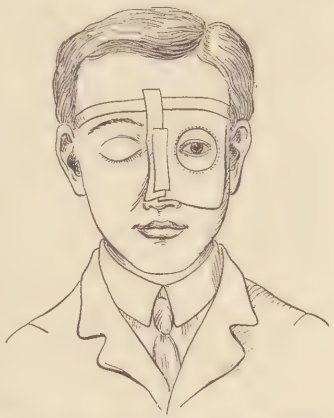


Fig. 103.—Application of Buller's shield.

The disease is almost invariably bilateral, is more common in men than women, and resembles a catarrhal conjunctivitis with some swelling of the mucous membrane and redness of the lids. Small ulcers of the cornea may form. According to Heerfordt, it may assume the form of phlyctenular conjunctivitis. Iritis, iridocyclitis, and uveitis may follow this inflammation, just as they are seen associated with gonorrheal rheumatism. *Endogenous gonorrheal keratitis*, occurring before or after arthritis, has also been described, but the etiologic relation of gonorrhea in this regard is doubtful (Elschnig). Relapses occasionally occur; the average duration of the disease is about two weeks. The diagnosis rests upon the presence of gonorrhea, the absence of the gonococcus, and the ordinary bacteria of conjunctivitis in the secretion.

The treatment of this affection demands the same remedies useful in ordinary conjunctivitis. Heerfordt advises massage with an ointment of yellow oxid of mercury and a lotion of copper sulphate (0.25 per cent.).

Non-specific Purulent Conjunctivitis.—Purulent conjunctivitis, not gonorrheal in origin, may be caused by the secretion of diphtheritic

[1] Care should be taken to provide a watch-glass of the ordinary form, not one with a concave center.

[2] Some surgeons apply the name *gonorrheal ophthalmia* to this affection, and reserve the term *gonorrheal conjunctivitis* for the disease which is caused by a specific urethral discharge.

conjunctivitis, and occasionally by granular lids. As has been noted, the manifestations of Koch-Weeks' bacillus conjunctivitis and pneumococcus conjunctivitis are not infrequently so violent and the secretion so profuse and purulent that they might be classified among purulent inflammations of the conjunctiva. (See also page 195.) It should further be remembered that a catarrhal conjunctivitis, by neglect or injudicious external applications—for example, poultices—may be aggravated into an inflammation in all particulars resembling gonorrheal conjunctivitis. Infection from the nares and pneumatic sinuses can also cause severe inflammation of the conjunctiva, associated with much purulent discharge. The treatment is exactly that which has been described in connection with other forms of acute conjunctivitis. The best results follow the free irrigation of the conjunctival sac with a saturated solution of boric acid or bichlorid of mercury (1 : 10,000), and the use of argyrol, protargol, and nitrate of silver in the usual manner. If properly treated, corneal complications are uncommon.

Croupous or Pseudomembranous Conjunctivitis (*Plastic, Membranous Conjunctivitis*).—Of this disease, two varieties may be considered. The *first* is an inflammation of the conjunctiva, characterized by a soft, usually painless swelling of the lids, a membranous exudation upon the conjunctiva, and a scanty, seropurulent discharge.

Causes.—The affection in its pure form is rare. It is never found among the newborn, and rarely among adults, the majority of cases occurring in early life—*i. e.*, between first half year and the seventh year.

The contagiousness of the disease has not been established; no definite cause is known, although some relation exists between this disorder, scrofula, and eczema. Patients affected may at the same time be suffering from a croupous inflammation of the respiratory tract. Some authors regard the affection as a mild diphtheria. Non-virulent Löffler bacilli, staphylococci, and diplococci have been found in the secretion. Croupous conjunctivitis may also be due to the pneumococcus, the diplobacillus, the Bacterium coli, and the Koch-Weeks bacillus. Influenzal bacillus membranous conjunctivitis has been described (see page 191).

Symptoms.—These begin with an acute conjunctivitis, succeeded by swelling of the lids, which remain soft and pliant, and usually not painful to the touch. In a few days there is a deposit of a characteristic false membrane composed of coagulated fibrin, rather translucent and porcelain-like in appearance, beginning upon the retrotarsal folds, coating the inner surfaces of the lids, but not invading the bulbar conjunctiva. It may readily be removed without loss of the conjunctival tissue, and shows beneath a granular and somewhat bleeding surface. It is quickly reproduced. The cornea, except in severe cases, escapes.

Healing takes place in from ten to thirty days, except in those instances where the membrane is formed again and again, and the course of the disease may continue for months and even years, constituting the *recurring form of* pseudomembranous conjunctivitis.

Diagnosis.—The disease may be confounded with conjunctivitis neonatorum and diphtheritic conjunctivitis. From the former it is distinguished by the absence of profuse purulent discharge and the age of the patient; from the latter, by the soft swelling of the lids, the superficial character of the membrane, and absence of virulent Klebs-Löffler bacilli.

Treatment.—This should include the application of ice compresses, frequent removal of the discharge with a solution of chlorid of sodium or chlorate of potash, and later the cautious use of nitrate of silver (Knapp), or of argyrol and protargol. As in many instances it is difficult to exclude diphtheritic infection, the administration of diphtheria antitoxin should be ordered in doubtful cases.

The *second* variety of membranous conjunctivitis is due to streptococci, is rapid in development, and is associated with swelling of the lids and much discharge, and may quickly destroy the cornea. It occurs in children in connection with measles, scarlet fever, and influenza; but, according to Morax, may appear independently of febrile complications and may accompany impetigo. The prognosis is most unfavorable not only to eyesight, but also to life. The disease is often mistaken for diphtheritic conjunctivitis (it is sometimes called *streptococcus diphtheria of the conjunctiva*). Microscopic examination will decide the diagnosis. The treatment already detailed is indicated. According to Axenfeld the use of streptococcal serum is to be recommended.

Membrane-forming conjunctivitis, not strictly to be classified with either of the foregoing types, has several times been referred to. Thus, membrane formation may be noted as an intercurrent condition in several varieties of conjunctivitis—for example, in gonorrheal conjunctivitis, pneumococcus and Weeks' bacillus conjunctivitis, trachoma, and exanthematous conjunctivitis. Membranes may also form in the conjunctiva as the result of chemical, mechanical, and thermic irritants. It is especially noteworthy after certain injuries of the conjunctiva—for instance, lime-burns.

Diphtheritic Conjunctivitis.—The deep-seated or necrotic variety of this disease is characterized by a board-like, very painful swelling of the lids, a scanty seropurulent or sanious discharge, and exudation within the layers of the tarsal conjunctiva, which spreads to the ocular conjunctiva, and by pressure destroys the nutrition of the cornea.

Causes.—In addition to the Klebs-Löffler bacilli, which cause the disease, other micro-organisms, for example, streptococci, staphylococci, and non-virulent xerosis bacilli, are usually present in the discharge (Uhthoff). The disease, which is contagious, may originate from a similar case, or arise in the course of a purulent conjunctivitis. It has occurred, though rarely, with conjunctivitis neonatorum. At times it appears in connection with eczema of the face and borders of the lid, and is an occasional accompaniment of some acute illness, for example, scarlet fever or measles, the diphtheritic type of the inflammation being ingrafted upon the conjunctiva. The disease has been seen during epidemics of diphtheria, and may be part of a process which

passes from the nose to the conjunctiva, or may be due to direct inoculation with the diphtheritic poison.

It is commonest between the ages of two and eight, and is unusual in young infants. In certain localities in the south of France and the north of Germany the disease was formerly frequent. It is usually stated that the disease is comparatively rare in America and England; but Sydney Stephenson records a percentage of 1.25. This author regards conjunctival diphtheria and croupous conjunctivitis as one and the same disease.

Symptoms.—The patches appear in a *discrete* or *confluent* form; the lids are swollen with a characteristic, painful, board-like hardness. The false membrane is of a dull, grayish appearance, and is torn off with difficulty, and carries with it parts of the conjunctiva. If the process is deep, the subjacent structure is pale, infiltrated, and when cut into may be anemic and lardaceous. If the diphtheritic inflammation has been ingrafted upon a case of purulent conjunctivitis, the abundant secretion ceases, or becomes irritating and sanious.

Sloughing of the cornea is almost inevitable in severe cases, and rapid destruction of this membrane may take place in twenty-four hours; even in mild cases ulcers may be expected.

Restlessness, fever, alimentary derangements, and nervous phenomena are usual constitutional disturbances, and the disease may be followed by loss of knee-jerk and paresis of various parts of the body. Albumin may be present in the urine, and occasionally diphtheritic conjunctivitis proves to be fatal (Stephenson).

Diagnosis and Prognosis.—This disorder is distinguished from the previous disease by the characteristic board-like infiltration of the lids and the bacteriologic examination, and has nothing in common with the flakes of false membrane sometimes seen in purulent conjunctivitis, from which it is further separated by the character of the discharge and by bacteriologic examination. The prognosis, as may have been inferred, is unfavorable, although with modern treatment more cases are cured without impairment of vision than formerly was possible. Occasionally, even if the diphtheritic membrane is only slightly developed, rapid necrosis of the cornea ensues.

Treatment.—The eyes should be frequently cleansed with warm boric acid solution or bichlorid of mercury solution (1 : 8000) and atropin drops should be instilled. Iodoform salve (or powder) may be freely applied within the conjunctival sac; indeed, vaselin itself is efficient in these circumstances.

Internally, quinin, iron, and mercury have been recommended; but the greatest reliance should be placed upon diphtheria antitoxin, which should be promptly administered exactly as it is in ordinary faucial diphtheria. At the first dose, 1500 to 2000 units of antitoxic serum may be injected into the lateral abdominal wall, and repeated in ten or twelve hours, according to the severity of the symptoms.

The sound eye may be covered with a bandage or Buller's shield. The patient should be isolated.

In addition to the deep-seated, necrotic variety of diphtheria of the conjunctiva, the disease, according to Uhthoff, Sourdille, Elschnig, and Morax, may assume a benign aspect and a superficial pseudomembranous form. Why virulent diphtheritic bacilli sometimes cause a superficial and sometimes a deep intersitial type of the affection has not, according to Uhthoff and Coppez, been determined. The former author also describes a simple catarrhal conjunctivitis in association with diphtheritic bacilli.

Phlyctenular Conjunctivitis (*Phlyctenular Ophthalmia; Scrofulous Ophthalmia; Eczema of the Conjunctiva*).—This is a form of inflammation of the conjunctiva, characterized by the appearance of one or more grayish elevations, situated chiefly upon its bulbar portion in the immediate vicinity of the cornea. Less frequently the phlyctenules appear upon the tarsal conjunctiva, those on the lower lid being of firmer consistence than those on the bulbar conjunctiva (Schiele).

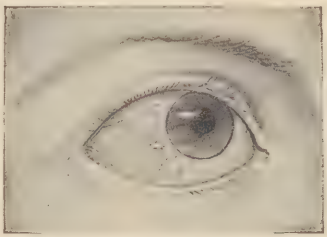


Fig. 104.—Phlyctenular conjunctivitis (Children's Hospital).

Causes.—The disease is believed to be of constitutional origin, and its subjects are often tuberculous and badly nourished children. Errors of diet, unwholesome foods, and the abuse of tea and coffee act as predisposing causes. It often follows the exanthemata, especially measles. Infectious rhinitis is always present, and usually the submaxillary and cervical glands are swollen, and there is eczema of the lip and nares. There is a distinct clinical association between this disease and eczema. It is possible that the active micro-organism is the *Staphylococcus pyogenes aureus* or *albus*, which is found beneath the epithelium of the affected conjunctiva, but an endogenous origin of the disease cannot be wholly excluded. That the disease is a true tuberculosis is disputed by Axenfeld.

Symptoms.—The disease occurs in a single and a multiple form; the pimples or phlyctenulæ lie near the corneal margin or directly upon it, and are usually from 1 to 3 mm. in diameter.

If the elevations are large, yellow, and contain purulent material, the disease has been called *pustular ophthalmia.*

In any circumstances it is accompanied by dread of light, injected blood-vessels, and increased lacrimation. The conjunctiva may be transparent, or the disorder associated with a mucopurulent conjunctivitis. After the exanthemata this association is common.

In the multiple form, numerous minute phlyctenules may be scattered over the entire conjunctiva, and are accompanied by decided general red injection, irritation, and photophobia. The disorder subsides in from ten days to two weeks.

Treatment.—Locally, mild antiseptic collyria, especially lotions of boric acid, are useful. Much irritation indicates atropin drops and the

occasional instillation of holocain to relieve the photophobia. The eyes may be protected by colored glasses.

After the acute symptoms have subsided the best results are obtained by introducing the yellow oxid of mercury, 1 grain to 1 dram (0.065–3.885 gm.), into the conjunctival sac or by dusting into it calomel, provided the patient is not taking iodid of potassium, otherwise a reaction between the potassium iodid in the tears and the calomel occurs, with the ultimate formation of double iodids, which are caustics (*calomel conjunctivitis*). Applications of borobismuth ointment to the nasal eczema are recommended by Schiele, who also speaks favorably of such antiseptic powders as gallicin, iodogallicin, and bismuth oxyiodid tannate. Linear cauterization of the fornix has been advised in severe cases.

An excellent regulation treatment is a mild course of mercurial laxatives. Simple diet, good air, exercise, and internally, quinin, iron, arsenic, and cod-liver oil, complete the therapeutic measures.

Phlyctenular conjunctivitis is so closely allied to phlyctenular keratitis that the separation of the two affections is purely artificial, and this account is a preface to the description of the more exact disposition and relation of the phlyctenules, which appears on page 242.

Vernal Conjunctivitis (*Fruehjahr's Catarrh*, Saemisch; *Phlyctæna Pallida*, Hirschberg; *Periodic Hyperplastic Conjunctivitis*, Wicherkiewicz; *Fibroma of the Limbus; Spring Conjunctivitis*).—This form of conjunctival disease is characterized by photophobia, stinging pain, considerable mucous secretion, the formation of flat granulations in the palpebral conjunctiva, and a hypertrophy of the conjunctiva surrounding the limbus of the cornea.

Causes.—Definite information in regard to the cause of this disease is lacking. It is possible that some specific micro-organism exists which has not yet been isolated. Although frequently the disorder returns in the early spring and subsides in the fall and winter, cases may occur in any month of the year, and its designation, *spring catarrh*, is not a good one, because the affection is in no sense a catarrhal one, and it does not necessarily occur in the spring. It is most frequent between the ages of five and fifteen, but occasionally occurs in advanced adult life and in very young children, even those but a few months of age. According to Posey's investigations, it is more frequent in males in the proportion of 85 to 15 per cent. It may accompany the disease known as hay-fever. Some writers decline to consider vernal conjunctivitis a distinct disease, but look upon it as a hypertrophic form of chronic conjunctivitis. In a few instances it appears to be hereditary, and Meyerhof describes a familial development of vernal conjunctivitis. At times there appears to be a congenital disposition to the disease.

Symptoms.—The affection begins like an ordinary conjunctivitis and is almost always bilateral; a few unilateral cases are on record. There are photophobia, more or less mucous secretion, circumscribed pericorneal injection, and the formation at the limbus of small, gray, semitransparent nodules, which swell up and overlap the edge of the cornea.

Three varieties of the disease are described—the limbus, palpebral, and mixed forms. The conjunctiva of the bulb is injected, that of the lids is slightly thickened and of a dull, pale color, as if brushed over with a thin layer of milk. This milky film seems to be the most constant symptom of the disease, and is to be observed before the granulations appear on the inner surface of the lids. When they appear in typical form, these granulations cover the tarsal conjunctiva, are flattened, and contain deep furrows between them. In the colored race there is a brownish pigmentation of the scleral base of the hypertrophied masses (Burnett).

The disease may be distinguished from trachoma by the flattened appearance of the granulations, the absence of infiltration and pannus, and the history of recurrence at special seasons of the year. Elschnig calls attention to an arrangement of the blood-vessels of the tarsal conjunctiva which he considers peculiar to spring catarrh. The normal vascular distribution is replaced by innumerable small vessels arising perpendicular to the conjunctival surface. Mixed forms of spring catarrh and trachoma have been described (May, Meyerhof).

The *pathologic histology* of the lesions has attracted much attention in recent years. According to Axenfeld, who has elaborately investigated this disease, the primary lesions arise in the subconjunctival tissue, followed by proliferation of the epithelium. An accumulation of plasma cells takes place, succeeded by a homogeneous sclerosis of the connective tissue. Elastic tissue is abundant. The milky appearance of the surface of the conjunctiva is due to subepithelial hyalin thickening. Eosinophils are frequent in the conjunctiva and occur in large numbers in the secretion. There is no essential difference between the tarsal and palpebral manifestations of the affection, unless future investigations should confirm the assertion of some observers that in the bulbar proliferations the epithelial changes take precedence.

The *prognosis* of the disorder is not unfavorable, except in so far as relapses are concerned, which make its course a long one, sometimes lasting from eight to ten years, or even longer. Usually the activity of the process begins to subside after it has existed for six or seven years. Slight opacity of the cornea may result.

Treatment.—During the height of the attack the eyes may be protected with dark or yellow-tinted glasses. Cold compresses afford some relief. Weak astringents and antiseptic lotions, such as have been recommended for ordinary conjunctivitis, are useful. The application of boroglycerid to the everted lid is sometimes valuable, and the systematic use of a preparation of suprarenal extract, or of adrenalin chlorid (1 : 10,000), to which a 1 per cent. solution of holocain is added, is of service. Axenfeld suggests the application of a solution of sulphate of quinin, and Elschnig advises the instillation of a watery solution of ichthyol (1 to 2 per cent.). Salicylic acid ointment (1 per cent.) is recommended, and massage with yellow oxid ointment may be tried. Electrolysis has been employed, and brossage has been advised by L. W. Fox. Exuberant granulations and limbus hypertrophies may be ex-

cised. According to Wicherkiewicz, a collyrium of antipyrin (10 per cent.), and instillations of 2 or 3 per cent. of protargol are efficient. Starr, in Buffalo, and Allport, in Chicago, have recommended the *x*-rays in the same manner as they are applied in trachoma, and have reported favorable results. Abbé and other surgeons have advised the application of radium, and have recorded notable successes. There is some evidence to show that the internal administration of arsenic is of advantage. Associated intranasal inflammation should be treated. Change from a warm to a cool climate is of service.

Follicular Conjunctivitis (*Follicular Ophthalmia; Conjunctivitis Follicularis Simplex; Trachoma Folliculare; Folliculosis; "School Follicles"*).—This affection is characterized by the presence of small pinkish prominences in the conjunctiva, for the most part in the retrotarsal folds, and usually arranged in parallel rows. The descriptive term "conjunctivitis" may be applied to the affection if the signs of inflammation are associated with it; if the latter are absent, the term *folliculosis of the conjunctiva* is more appropriate.

Causes.—The disease arises under the influence of poor hygienic surroundings, especially in pauper schools, where it may appear as an aggravated epidemic, but it is frequently seen in mild form, especially among children during their school years, particularly if they are the subjects of anemia and chlorosis. Indeed, in so large a percentage of school children can tumefaction of the conjunctival lymph-follicles be found that the name *school-folliculosis* has been suggested by Greeff. Evidently refractive errors are the exciting cause of many of these cases. Enlargement of the follicles may also be caused by local irritants and some medicaments—for example, atropin (see page 228).

Much difference of opinion exists as to whether folliculosis should be placed in a separate category from granular conjunctivitis, or whether it should be regarded as an early stage of the latter disease. Although transitional forms apparently exist, the evidence, clinically at least, warrants the belief that this affection is distinct from granular lids, because folliculosis occurs where trachoma is unknown, and because the follicles disappear without leaving a trace of their existence or producing scar tissue in the conjunctiva. Histologically, however, there is no decisive difference between fresh follicles and fresh trachoma bodies. It would seem, as Greeff insists, that folliculosis may arise under the influence of various excitants, and in this sense is a symptom and not a distinct disease.

Symptoms.—The children—for it mostly occurs in children and young people—complain of slight dread of light and inability to continue at close work, and inspection reveals numerous round elevations in the conjunctiva, chiefly along the fornix, which are tumefied lymphatic follicles. The color of the follicles varies from nearly white to a decided pink. After their disappearance the conjunctiva regains its natural state.

If with the enlarged follicles inflammatory symptoms are combined, the disease is a true *follicular conjunctivitis;* the lids are swollen,

reddened, and their margins streaked with secretion, which, at first thin, may become more purulent and quite abundant. In certain circumstances the disease resembles the condition termed "swelling with catarrh" (see page 194), except that the development of the follicles is much more evident. The inflammatory form of the affection may assume a more *chronic type*, with special development of the follicles in the fornices, and only secretion enough to stick together the lids in the morning.

The disorder is to be distinguished from granular lids by observing that the small bodies are neither so large as true granulations nor so highly colored as hypertrophied papillæ; that the mucous membrane is not affected more deeply than the lymphatic follicles; and that cicatricial changes are not present.

The *prognosis* is good in so far as the fate of the mucous membrane is concerned, but the disorder is troublesome and will often last for months, and, under imperfect hygienic surroundings and in crowded asylums, may prove to be a stubborn affection.

Treatment.—Locally, boric acid, either alone or made up with a few minims of alcohol to one ounce of water, weak bichlorid solutions, and occasional applications of boroglycerid or tannin and glycerin are useful. A salve of $\frac{1}{2}$ grain (0.0324 gm.) of sulphate of copper to the dram (3.885 gm.) of vaselin has been highly extolled. If there is much secretion, the usual treatment of conjunctivitis is required, especially silver and its various salts.

Refractive error should be corrected because ametropia aggravates the disorder. In stubborn cases, especially in asylums, expression of the swollen follicles with suitable forceps should be performed (see page 617).

Granular Conjunctivitis (*Granular Lids; Trachoma; Egyptian Ophthalmia; Military Ophthalmia*).—This is a disease of the conjunctiva in which this membrane loses its smooth surface, owing, in the language of Saemisch, to an inflammatory infiltration of its adenoid layer, associated with the development of follicles ("granulations") and enlargement of the so-called papillary layer. After absorption and metamorphosis of the inflammatory material, cicatricial changes are found.

Causes.—Formerly it was the custom to separate this disease into two forms—*acute granulations*, or *acute granular conjunctivitis*, and *chronic granulations*, or *chronic granular conjunctivitis*, and certain systematic writers—for example, Saemisch—continue this distinction. In so far as the author's observations are concerned, they lead him to agree with those who maintain that the so-called acute trachoma, at least in the majority of cases, represents an admixture of ordinary granular lids and acute conjunctival catarrh. An attack of acute conjunctivitis may precede the development of trachoma. According to MacCallan, in Egypt, the acute symptoms are most often caused by the Morax-Axenfeld bacillus, the Koch-Weeks bacillus, and the gonococcus. Greeff, however, seems to have proved by inoculation that there is an acute trachoma which may develop in a few days.

Granular conjunctivitis may arise apparently under the influence of poor hygienic surroundings, and in institutions where the inmates are crowded together the disease may readily spread.

It has been stated that certain individuals, especially of lymphatic constitution, are predisposed to granular lids, but, although its subjects are often pale and anemic, because they live in badly ventilated homes, there is no known constitutional disorder which causes the disease, as it may attack those who are in perfect health. Personal susceptibility to the disease varies. Racial predisposition has been maintained, the Mongolian race being especially liable to the affection; but, according to Axenfeld and Boldt, marked racial predisposition is doubtful. In Egypt and the eastern countries an admixture of trachoma and purulent ophthalmia is very prevalent. Trachoma is generalized throughout Egypt, affecting about 95 per cent. of the population (MacCallan). According to MacCallan, children in Egypt are usually infected during the first two years of life. It may develop in the newborn as the result of infection during delivery, inasmuch as trachoma of the genital passage occurs. It is exceedingly common among the poorer classes in Ireland and Russia, and Jews of the lower social grades are very prone to be affected. The Indians of our own country are frequently attacked, while pure negroes are almost exempt. The exemption of the negro, however, is denied by Minor. Trachoma is widely distributed through certain regions in the Middle West and western portions of the United States.

Among the immigrants to our country—Poles, Jews, Russians, Hungarians, and Italians—the disease is common, and occasions much anxiety among those whose duty is concerned with the inspection of these foreigners. The determining factor, however, is not so much connected with racial predisposition as with the uncleanly habits and unhygienic surroundings of those who are the subjects of the affection. The geographic distribution of trachoma has attracted much attention, and it has been found that the dwellers in certain regions of the earth where the climate is damp are readily affected, while it has been maintained that an altitude of 1000 feet confers comparative immunity from the disease and facilitates its cure. Trachoma, however, occurs in mountainous regions, for example, in our own country among the Indians as well as among white settlers.[1] A climatic predisposition is doubtful.

Bacteriology of Trachoma.—Transference of the morbid material from a trachomatous conjunctiva to another eye may result not only in a purulent conjunctivitis, but in a disease like the one from which it came. In this sense the disease is specifically contagious, although the affection is not conspicuously contagious by secretion inoculation (Axenfeld). There is no proof that trachoma is caused by microorganisms of the bacterial group or by blastomycetes.

Halberstädter and Von Prowazek, working in Java, and Greeff,

[1] According to Burnett, trachoma occurs at an altitude of 4700 feet. Van Millingen denies the influence of altitude and an immunity for certain races.

Frosch, and Clausen, in Germany, discovered in the discharge and follicle contents of trachoma very small granules, resembling diplobacteria. They are surrounded by a zone (hence called by Prowazek *chlamydozoa*), and occur either isolated or grouped together within the cell next to the nucleus (see also page 203). They increase in number, and gradually occupy almost the entire protoplasm of the cell which is destroyed, and they are set free in the secretion. Certain small bodies are seen in the protoplasm of the cells and outside of the cells, and they almost always accompany the "inclusions," and are called "Lindner's initial bodies." The Prowazek bodies are found in fresh, untreated trachoma, less easily in granular lids of long standing. Their exact nature is unknown, although, according to Axenfeld, they probably are neither cell-products, metamorphosed gonococci (Herzog), nor the products of mucoid degeneration. They are so seldom present in other conjunctival disease that their detection is highly significant. Their absence, however, does not exclude trachoma. They are usually

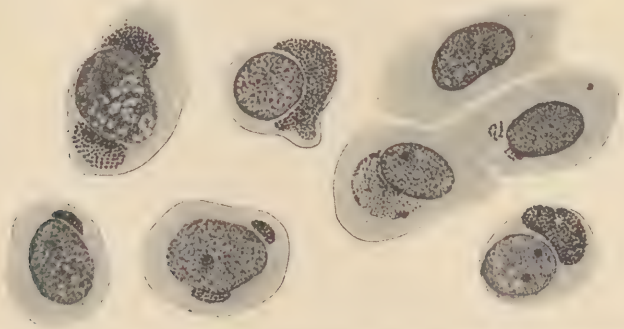


Fig. 105.—So-called trachoma bodies—epithelial inclusions (Axenfeld).

known as "Prowazek or Prowazek-Halberstädter bodies or corpuscles," and they have also been found in ophthalmia neonatorum (Heymann) (see page 203), in their early stage in the normal conjunctiva, and in some forms of chronic conjunctivitis (Erdmann).

Pathology and Varieties of Trachoma.—The pathognomonic appearance and essential element of the disease trachoma are the "granulations," or "trachoma bodies," or follicles.[1]

Two views have been held—the one that the trachoma bodies have a special pathologic character; the other that they are derived from the lymphatic follicles, which, although poorly developed, are probably present in the natural human conjunctiva, and some authors declare that these follicles and their changes originate all the anatomic and clinical qualities of trachoma. Although it may not be possible to distinguish in the early stages trachoma bodies from enlarged lymphatic

[1] It should be remembered that the word "granulations" refers to the characteristic feature of trachoma, and not to surface granulations which may form during the course of the disease. The Prowazek bodies are also called "trachoma bodies" by some authors, for example, by Greeff.

follicles, there is a difference in the nature of the two conditions, and for the most part the "unitarian standpoint" with reference to trachoma has been abandoned—that is, the theory that all follicles in the conjunctiva represent trachoma. Certainly so-called "benign follicles" exist which disappear without a residue of lesions (see page 214), and, on the other hand, an infection with follicular formation arises which subsides after a long period of time and leaves cicatrices (see page 219). Between these two forms are the "border-line" cases which are difficult to classify. The onset of trachoma may be preceded by a Koch-Weeks bacillus conjunctivitis in which Prowazek bodies can be found. Some authors deny that trachoma is a specific disease, believing that it is the pathologic expression of the reaction of the conjunctiva to various irritants (Walker).

Fig. 106.—Trachoma of the retrotarsal fold: *a*, Follicle; *b*, diffuse infiltration; *c*, Henle's gland with goblet-cells; *d*, lymph-vessel filled with leukocytes (× 30) (Holden).

The following varieties of chronic trachoma have been recognized by systematic writers:

1. *Papillary trachoma*, in which the trachoma bodies or follicles are sparsely present and hidden from view by hypertrophied conjunctival papillæ, or, more accurately, pseudopapillæ. The blood-vessels are enlarged, and there is marked increase in the number of lymphoid cells. The development of the follicles in the adenoid layer lifts above them the thickened epithelium. This form is sometimes spoken of as *chronic trachoma*.

2. *Follicular trachoma*, in which the presence of the "follicles" or trachoma bodies is the chief characteristic. These bodies are round collections of lymphoid cells which may possess an incomplete capsule, and which, as before stated, are elaborately developed in the adenoid layer of the conjunctiva. Some authors consider follicular conjunctivitis (see page 214) a variety of this type. Systematic writers have differentiated the following cellular elements in the trachoma follicle: lymphocytes, which form the chief constituent of the peripheral zone; mononuclear leukocytes, which compose the chief portion of the follicle; phagocytes, which are found among the leukocytes; and certain acces-

sory elements—for example, multinuclear cells, etc. Beneath the follicles are dilated lymph-vessels, and blood-vessels may extend into the follicles. The lymphadenoid tissue surrounding the follicles is infiltrated with leukocytes. Some of the cells of the follicles are discharged or absorbed; others are converted into connective-tissue fibers, which, by their contraction, produce the changes described on page 220. According to Parsons, the invariable termination of trachoma in cicatrization is brought about by absorption of the contents of the follicles and proliferation of the connective tissue of the conjunctiva, it being doubtful if the elements of the follicle can themselves form fibrous tissue.

In one form, designated by Knapp *non-inflammatory follicular trachoma*, the spawn-like granulations develop in the conjunctiva without evidence of inflammation, and have been regarded as analogous to nasopharyngeal adenoid hypertrophies.

3. *Mixed trachoma*, in which the follicles or bodies lie among hypertrophied and inflamed papillæ, but are not hidden by them. This type is sometimes described as *diffuse* or *complicated trachoma*.

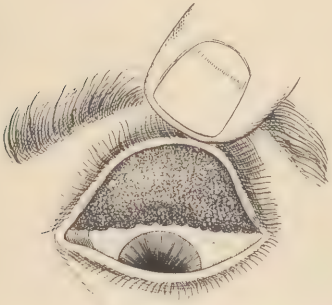


Fig. 107.—Follicular trachoma (Johnson).

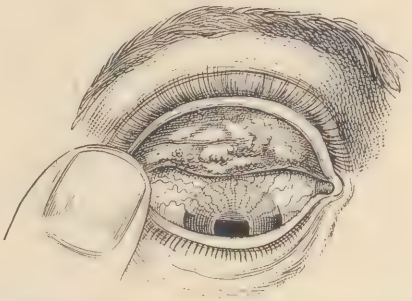


Fig. 108.—Typical granular lid and beginning cicatrization, with pannus (Berry).

4. *Sclerosing trachoma* (Knapp), in which, after an initial stage of ordinary granulations, leathery (fibrous), flattened excrescences develop in the upper tarsal and retrotarsal conjunctiva.

5. *Cicatricial trachoma*, in which atrophy and scar tissue are manifest—"the end stage of uncured cases" (Knapp).

Although the separation of granular lids into these varieties is convenient from the clinical standpoint, such a separation cannot be maintained on histologic grounds. Indeed, Saemisch maintains that the terms "papillary" and "follicular trachoma" should be avoided, as the first corresponds with blennorrhea, and the second with follicular conjunctivitis. He also deprecates the name "mixed trachoma," and believes the disease should be divided into *simple granular conjunctivitis* and *granular conjunctivitis in the stage of cicatrization*.

Symptoms.—The "granulations or follicles" often appear without antecedent inflammation, and so insidiously that their real nature is for a time unknown to the patient. They usually develop in the form of

grayish-white, semitransparent bodies, which vary in size according to their stage of development, and which, from fancied resemblances, have been called "sago-grain" or "vesicular" granulations. They may be disseminated or arranged in parallel rows, and have sometimes been likened to the appearance of frog's spawn (follicular trachoma). The granulations are, for the most part, confined to the palpebral conjunctiva, and the upper retrotarsal fold, which is a favorite location, should be well exposed during the examination. Occasionally granulations are found upon the caruncle and semilunar folds.

The mucous membrane is pale or yellowish red, unevenly rough, and contains the trachoma bodies, or follicles, which have a more or less deep situation and fill up the tissue. If they have not followed an acute process, there are few or no irritative manifestations and little discharge—perhaps only sufficient to glue together the lids. As time goes on the closely packed masses compress the true conjunctival tissue and its circulation, and a superficial vascularity of the cornea may appear. This stage may last for months and be subject to numerous variations.

In the next stage vascularity is increased, the follicles grow larger, soften, and their contents are forced out by the pressure of the surrounding infiltrations, forming, in association with the hypertrophied conjunctival papillæ, red protuberances (hypertrophied pseudopapillæ). This period is associated with strong irritation and mucopurulent or purulent secretion, photophobia, local pain, and corneal complications.

During the time of fatty degeneration and softening, which by some authorities is deemed a process of ulceration, fresh follicular (granular) eruptions take place, in turn to go through the same changes which their forerunners have undergone. The mucous membrane now has a flesh-red appearance; it is with difficulty that the "granulations" are distinguished from the papillæ, and, indeed, they are united with them, forming variously shaped diffuse or isolated protuberances. Sometimes resorption of the follicles, which can take place at any stage, appears to occur by retrogression without softening. Usually degenerative changes in the cells take place. Fusion and softening of the closely packed follicles may be followed by hyaline degeneration, giving rise to a gelatinous appearance, which is sometimes designated *gelatinous trachoma.*

In the final stage cicatrization begins, and gray-white scar-lines appear, intersecting the remains of the old "granulations." When these cicatrices lie parallel to the ciliary borders, they present, on eversion of the lid, a typical appearance[1] (Fig. 108).

By a gradual process of cicatrization of the old "granulations" and successive new crops, a chronic induration and diffuse scar tissue results (cicatricial trachoma). This being firmly attached to the tarsus, which itself has undergone softening through a lymphoid infil-

[1] It is convenient to thus divide the disease into three stages, as Raehlmann has done, but it is not always possible to separate sharply each stage by symptoms or appearances peculiar to itself.

tration, contracts, and the deformities of the lid and its border, so common in this disease, result. The fibroid induration of the mucous membrane affects all portions, and there may be almost entire obliteration of the conjunctival sulcus, or the membrane may undergo a species of drying up, to which the name *xerosis* has been applied. Individuals with granular lids, in the stage of thickening of the mucous membrane, have an almost characteristic sleepy look, peering uncertainly through narrowed palpebral fissures, caused by the ptosis-like droop of their indurated eyelids.

In so-called *acute granular conjunctivitis* the lids are swollen, the conjunctiva reddened, the conjunctival pseudopapillæ hypertrophied and elevated by the underlying lymphoid infiltration, while between are found the yellowish, round "granulations." The dread of light is intense, and in forcible separation of the lids scalding tears gush out, and later mucopurulent discharge appears. Still later, vascularization and ulceration of the cornea may develop. As before stated, many authors are disinclined to regard these manifestations as a special form of trachoma, but consider them to be a mixture of granular lids and catarrh; acute exacerbations of chronic trachoma are common.

Sequelæ of Granular Lids.—The most important results of long-standing granular lids are trichiasis, distichiasis, and entropion, conditions already described (see page 181), atrophy and shrinking of the conjunctiva from cicatricial changes (see page 230), cloudiness and ulceration of the cornea, and *pannus*.

Pannus may be looked upon as a form of *vascular keratitis*, which always begins in that portion of the cornea covered by the upper lid, but which, in severe cases, may involve its entire surface. It depends upon the formation of new blood-vessels between the corneal epithelium and Bowman's membrane, associated with collections of round cells. It may be composed of only a few vessels (*pannus tenuis*), or be thick, fleshy (*pannus crassus*), and bulging in appearance. If softening and ulceration occur, the true corneal tissue is invaded.

It is sometimes taught that pannus is mechanical in origin and is caused by the friction of the granulations. According to Raehlmann, however, it should not be considered as a simple traumatic irritation, but as a follicular process, with the formation of lymphoid infiltration, analogous to the same pathologic condition in the conjunctiva; in short, pannus is to be regarded as a special implantation of the trachoma process in the layers of the cornea. Mutermilch believes that pannus does not depend upon roughness of the conjunctiva, but upon the destruction of living epithelium, and where this is regenerated, the pannus subsides. Extensive and deep ulceration may complicate pannus, which, in turn, may lead to the development of iritis; or the cornea may become entirely opaque; or, finally, the ulceration may be followed by perforation of this membrane and staphylomatous bulging.

Diagnosis.—This usually presents no difficulties. An examination of fresh material should reveal the Prowazek-Greeff granules, but, as has already been pointed out, the presence of these bodies, while highly

significant, is not sufficient to establish a diagnosis; *i. e.*, their absence does not exclude trachoma. The disease is made evident by direct inspection of the everted lids unless the associated swelling of the papillæ is so great as to obscure the "granulations," especially in the forms of papillary trachoma. Hypertrophied conjunctival papillæ, chronic blennorrhea, and surface granulations must not be mistaken for trachoma. The clinical distinctions existing between trachoma and follicular conjunctivitis have been pointed out. Great care should be exercised in the inspection of immigrants, and those undoubtedly trachomatous should be deported. All suspected persons, and all those in whom the diagnosis is uncertain, should be isolated and detained until the exact nature of their conjunctival trouble is ascertained.

Prognosis.—In the best circumstances, granular lids, when well established, is a tedious disease, and greatly endangers the vision of the patient. Relapses are frequent, and at any time the disorder is likely to assume an intense inflammatory action. Its contagious character renders the affection especially dangerous in schools and in any institution where large numbers of inmates are gathered together. The discharge, even when present in slight degree, is readily conveyed from one subject to another by the careless use of towels and common utensils. Great caution is necessary in such circumstances to prevent a disastrous epidemic.

Treatment.—The treatment of chronic granular lids includes the application of caustics, astringents, antiseptics, and certain so-called specific remedies, operative procedures, and general medication.

Local applications of astringent and caustic preparations are used to cause absorption of the "granulations," but these should not be of such strength as to produce cicatricial changes more harmful than the original malady.

A variety of substances has been employed; indeed, it is safe to assert that there is scarcely an antiseptic or caustic agent the use of which is permissible in ocular disorders that has not been tried in the effort to alleviate the symptoms of this disease. If granular conjunctivitis is associated with much discharge in the sense of mucopurulent secretions, the ordinary antiseptic and slightly astringent lotions are useful, and should be freely employed to irrigate the conjunctival culdesac. Those which serve the best purpose are saturated solutions of boric acid, bichlorid of mercury (1 : 5000 or 1 : 10,000), and cyanid of mercury (1 : 1500).

Formerly, during the stage of conspicuous lymphoid infiltration and decided follicular eruption, without the presence of much discharge, the direct application to the everted lids of strong solutions of bichlorid of mercury (1 : 300 or 1 : 500) were much employed, and at one time the author was impressed with their value, but in recent years has practically discontinued them.

In the stage of softening of the granulations and swelling of the conjunctival pseudopapillæ, associated with mucopurulent and purulent

discharge, in addition to flushings with the antiseptic solutions already mentioned, nitrate of silver is of value, employed in the manner already described (see page 201). Generally it is not necessary to use a solution stronger than 2 per cent. Instead of nitrate of silver, argyrol and protargol in the usual strengths may be employed. In this stage solutions of permanganate of potassium (1 : 3000 and 1 : 5000) have been advocated, while stronger solutions (1 : 1000) have been applied directly to the everted lid.

At one time sulphate of copper was almost universally employed in the treatment of trachoma, and the author is convinced that it still occupies a useful place in the management of this affection. Where the eruption of new granulations is associated with beginning cicatricial metamorphoses of old crops and their surrounding tissue, this remedy is of value. The crystal of sulphate of copper should be smooth and carefully applied to all portions of the affected areas, especially to the retrotarsal folds, and the treatment followed by washing the surface with cold water. It is a painful remedy, and in sensitive patients there is no objection to cocainizing the eye. In order to render sulphate of copper painless, it has been suggested to fuse it into a crayon composed of this drug, orthoform, holocain, and gum tragacanth. Sulphate of copper, dissolved in glycerin (5 per cent. solution) is a most useful application.

In place of sulphate of copper, copper citrate (cuprocitrol), originally recommended by F. R. Von Arlt, has found favor with some surgeons. It may be employed in a 5 or 10 per cent. ointment, which is introduced well in the conjunctival sac, and gentle but thorough massage used immediately afterward.

During the later stages of trachoma, in order to hasten the absorption of remaining granulations and perhaps to prevent the tendency to xerosis, boroglycerid (30–50 per cent.) is a useful remedy, applied in the usual manner with a cotton mop. In mild cases, or after an impression has been made with stronger caustics, a favorite astringent is tannin and glycerin, 30 to 60 grains to 1 ounce (1.95–3.9 gm. to 30 c.c.), or the everted lids may be touched with an alum crystal. Among the many additional remedies which have been tried in this affection the following may be mentioned: Liquid carbolic acid, liquor potassa, beta-naphthol, hydrastin, iodoform, or aristol (in powder or salve), an ointment of the yellow oxid of mercury, calomel, iodid of silver, ichthargan (2 to 3 per cent.), itrol in powder, ichthyol, and cyanid of mercury (1 : 500), which is energetically rubbed by means of a tampon of cotton wool over the granular surface.

Granular lids are liable at any time to develop acute symptoms: increased discharge; exacerbation of pannus, with clouding and ulceration of the cornea; hyperemia of the iris, and acute pain in the brow and temple. Usually severe local applications must be discontinued, and the treatment instituted which is applicable to acute conjunctivitis, and which need not be here repeated. Hot compresses are often agreeable, and the pupils should be dilated with a solution of atropin or

scopolamin. In this stage and, indeed, in other stages, especially if pannus is present, it would seem that dionin is of some value.

The *x-ray treatment of trachoma* has occupied a very large share of attention within the last few years, and has been recommended by Mayou, Stephenson, Walsh, and other surgeons.

The author's experience with this method of treatment is too limited to render an expression of opinion from him of value. In the few cases in which he has used it and seen it employed the results were indifferent, certainly not any better than those obtained by ordinary therapeutic agents or operative procedure. With improved technic, and particularly with the ability to measure the exact dosage of the rays, it would seem that the method offers a fair prospect of success, and should be given the fullest trial. Radium has also been used in the treatment of trachoma, but Charles H. May, who has studied the action of this substance in this respect, concludes that the results obtained are not so favorable as those secured with sulphate of copper. Carbon-dioxid snow has also been employed; applications for a few seconds once a week at first, later the time may be increased to twenty seconds.

Stephenson and Walsh also recommend the application of the *high-frequency current* through a vulcanite electrode applied to the upper lid in the treatment of severe trachoma.

Operative Procedures.—These include the various methods for removing the granulations: Scarification of the conjunctiva; abscission of the granulation; excision of the retrotarsal fold or of a strip of the infiltrated fornix; removal of a part of the tarsal conjunctiva at the same time that the strip of infiltrated fornix is excised (the so-called combined excision); extirpation of the tarsus (Kuhnt's extirpation) and squeezing or rolling out the trachoma follicles with suitable instruments, especially with Noyes', Knapp's, or Kuhnt's forceps. Removal of the granulations by means of a curet or stiff brush, and then rubbing into them strong solutions of bichlorid of mercury or cyanid of mercury (*grattage, brossage*), are measures that have been much employed. Grattage is performed by D. H. Coover with strips of sterilized sand-paper. The methods of applying the various operative procedures are described on page 617.

Of the methods just enumerated, expression of the follicles with suitable forceps, particularly in the so-called follicular forms of trachoma, is the most satisfactory, although both simple and combined excision of the infiltrated fornix sometimes yields exceedingly satisfactory results. Some surgeons, notably Mr. George Lindsay Johnson, recommend electrolysis in the treatment of trachoma.

Treatment of Trachoma with Pannus.—If the pannus is limited in degree, it requires no special treatment, as it will disappear with the absorption of the granulations; but if it is extensive, and especially if associated with ulceration, special treatment should be directed toward its cure. This includes the local remedies which are appropriate for a vascular keratitis, namely, an antiseptic lotion, the various mydriatics, and occasionally dionin.

Inveterate pannus, without ulceration of the cornea, at one time was treated by the production of a violent conjunctivitis, characterized by the formation of a somewhat clinging false membrane, with a 3 per cent. infusion of *jequirity*, painted upon the everted lids. This method was introduced by de Wecker to substitute the old-fashioned inoculation of the conjunctiva with blennorrheic pus. It has also been advised to apply the same drug in fine powder, a little at a time, exactly upon the portions of the granulations to be absorbed. The author has not been favorably impressed with jequirity. It has been recommended in this country by Cheatham and Sweet in recent years.

Since the introduction into ophthalmic practice of *jequiritol* and *jequiritol serum* by Roemer, these substances have been much employed in the treatment of trachoma. Jequiritol is an extract made from the seed of the *abrus precatorius*. It is used in a sterile solution mixed with 50 per cent. glycerin, so that an exact dose can be given without evil effects, which was not possible with abrin or the old infusion. According to Casey A. Wood, who has published directions for its application according to Roemer's method, it should be used as follows: The drug comes in four different degrees of strength. A single drop of No. 1 is instilled into the conjunctival sac by means of a capillary tube. If no severe reaction follows, the dose must be daily increased until a typical jequiritol inflammation develops. Occasionally no reaction occurs until No. 2 is used, when the acute inflammation is finally induced, and subsides in a few days. Subsequently the eye will endure a still stronger dose. Immunity is obtained after a number of inflammatory attacks, and at last the strongest dose produces no effect. If twenty-four hours after the employment of a dose the inflammation appears to be too severe, several drops of the jequiritol serum should be instilled into the eye frequently during the day. According to Hoor, jequiritol is indicated in old trachomatous pannus with cicatrized and degenerated conjunctiva. It is contraindicated in purulent processes of the cornea, in recent opacities, and in fresh trachomatous pannus. In spite of all care certain complications may arise—for example, edema of the lids, pain, facial eczema, and suppuration of the lacrimal sac. Evidently jequiritol is not without danger, and should be restricted to the cases already described.

The operation of *peritomy*, which consists of an excision of a ring of conjunctival tissue surrounding the cornea, has been much practised for the relief of severe pannus. Another method is to scrape away the opaque and vascular areas in the cornea with a small knife (Gruening). If the palpebral fissure becomes contracted by cicatricial changes, or if during inflammatory periods in trachoma the lids dangerously compress the cornea, the operation of *canthoplasty* affords relief.

General Medication.—It is a mistake to depend solely upon local measures for the relief of granular conjunctivitis, for, although the disease has no proved consitutional origin, its subjects give frequent evidence of malnutrition, and are sometimes affected with tuberculosis. Hence, in addition to every advantage that fresh air—if possible, at a

high elevation—good food, and pleasant hygienic surroundings can give them, iron, cod-liver oil, hypophosphite of lime, arsenic, and, in short, a general tonic regimen should be ordered. Suitable attention to the alimentary tract is important.

Parinaud's Conjunctivitis (*Infectious Conjunctivitis; Septic Conjunctivitis; Lymphoma of the Conjunctiva* [Goldzieher]).—This rather rare form of conjunctival affection was first accurately described by Parinaud in 1889, and has in recent times been the subject of very extended researches, particularly by Chaillous in France, and Gifford, Verhoeff, and G. S. Derby in this country. According to the last-named authors, the disease has been observed only in the temperate zone, and occurs a little more frequently in the autumn than at other seasons. The sexes are about equally affected, and all ages seem liable to it, the youngest patient recorded being one and a half years of age and the oldest fifty-nine. More commonly it is a unilateral than a bilateral disease; indeed, only a very few times have both eyes been affected.

The chief symptoms are the following: Swelling of the lid, usually most marked in the upper lid, hyperemia and edema of the bulbar conjunctiva, and a moderate mucopurulent discharge. The characteristic conjunctival lesions consist of large, reddish, semitransparent polypoid vegetations, small yellowish granules, erosions, and superficial ulcers. Sometimes the conjunctival growths are pedunculated. Very rarely corneal changes in the form of keratitis have been described. Glandular involvement usually takes place simultaneously with or very soon after the development of the ocular disease. In a few instances it has preceded them. Most often the preauricular glands are affected; more rarely the retromaxillary, the parotid, the submaxillary, and the cervical. Occasionally acute tonsillitis has been noted. The disease may last from one to five months.

So far, investigations have failed to isolate any of the known microorganisms as a causative agent of this disease, although McCrae found in one case a bacillus which resembled the Klebs-Löffler bacillus, which he regarded as the probable excitant of the ocular inflammation. Sinclair and Shennan have isolated two varieties of white staphylococci from the necrotic areas in one case. Parinaud believed that the disease was of animal origin, and Hoor maintains that in the majority of cases there is a history of an opportunity of animal contagion; but Verhoeff and Derby regard this theory, at present at least, as unsubstantiated. Recently Verhoeff has found in the lesions of this affection a filamentous organism classified as a *leptothrix*, which he regards as the cause of the disease. There may be difficulty in distinguishing the disease from tuberculosis of the conjunctiva, and to reach a definite diagnosis it may be necessary to inoculate the anterior chamber of a rabbit's eye with a fragment of the suspected tissue. Microscopic examination of the excised tissue reveals cellular infiltration, consisting of lymphoid and phagocytic cells and marked cellular necrosis.

The *treatment* recommended includes the ordinary antiseptic col-

lyria, nitrate of silver, or the newer silver salts, applications of sulphate of copper, and excision of the granulations. The injection of antidiphtheritic serum has been tried, and Sinclair and Shennan have instituted vaccine treatment, but the patient did not remain under observation for a sufficient time to demonstrate the value of the method.

Sporotrichosis of the conjunctiva has been reported by Morax, Cruchandeau, Gifford, and others. Thickening of the lid, nodular swelling of the conjunctiva, superficial yellowish ulcers, and adenopathy were present. (See also page 168.)

Chronic conjunctivitis (*chronic ophthalmia*), the result of an acute blennorrhea, has been referred to on page 206.

As an independent disorder, and assuming more the type of a hyperemia, it is a common disease in elderly persons. There are hyperemia, thickening of the papillary layer of the tarsal conjunctiva, swelling of the caruncle, soreness of the edges of the lids, and slight mucopurulent discharge. Often the bulbar conjunctiva is notably injected. Chronic conjunctivitis due to hypersecretion of the Meibomian glands (*conjunctivitis meibomiana*) and to "insufficiency of the eyelids," so that they close only with effort and remain open during sleep, is described by Elschnig. The latter may result in a form of xerosis of the conjunctiva (*tyloma conjunctivæ*, Saemisch). A chronic conjunctivitis of moderate severity with only slight injection and traces of abnormal secretion in the commissural angles has been called *conjunctivitis sicca*, and is especially aggravating on awakening. It is to this affection that the so-called "morning ptosis" is usually due, the patient being unable to open the eyes except with the help of the fingers, which elevate the lid. The relation of the diplobacillus to chronic and subacute conjunctivitis has been described (see page 192; see also Hyperemia of the Conjunctiva, page 187). In association with chronic conjunctivitis and chronic ciliary blepharitis delicate *flame-shaped marginal keratitis* may arise, the lesions having their bases at the limbus and their apices advanced about one-third way across the cornea (W. T. Holmes Spicer).

Treatment.—Cleanliness, with antiseptic lotions, the application of "lapis divinus," an alum crystal, or glycerol of tannin, gr. x to f℥j (0.65 gm. to 30 c.c.), are useful local measures. Aqueous solutions of suprarenal extract (8 per cent.) or adrenalin chlorid (1 : 10,000) will temporarily dissipate the congestion, but the author has not been able to persuade himself that they are curative in their action. The puncta lachrymalia should be examined, and if they are closed, they should be dilated and the lacrimal passages irrigated with an Anel syringe, and the nasal chambers should be carefully treated. Refractive error, which may keep up congestion, requires correction. For conjunctivitis meibomiana emptying of the Meibomian glands is recommended (Elschnig, Fridenberg). Boric acid in lanolin (2 per cent.) is useful if the conjunctival surface is too dry. If the Morax-Axenfeld bacillus is present, solutions of zinc sulphate or chlorid should be used (see page 194).

Egyptian and **military conjunctivitis** are terms which have at different times been loosely used to describe all forms of conjunctival inflammations occurring in crowded barracks and similar institutions, which assumed an epidemic tendency, pursued a more or less chronic course, and hence included varieties of acute and chronic blennorrhea and mucopurulent conjunctivitis, in addition to those cases which possessed as a fundamental diagnostic symptom "granulations" of the conjunctiva, and which eventuated in the formation of cicatrices.

Lacrimal conjunctivitis is really a form of chronic conjunctivitis depending upon obstruction of the lacrimal passages and the frequently associated blepharitis, and in the discharge of which *streptococci* are found. The eyelids are inflamed upon their borders, the cilia gathered in little tufts by the formation of small pustules at their bases, the conjunctiva is injected and tear-soaked, and there is a somewhat gummy discharge. This form of conjunctivitis may be complicated, according to Parinaud, with hypopyon and iridocyclitis.

The *treatment* requires that the lacrimal passages shall be rendered patulous, in addition to the ordinary remedies suitable for chronic conjunctivitis and ulcerated blepharitis.

Lithiasis conjunctivæ is a troublesome condition caused by a calcareous degeneration of inspissated secretion in the acini of Meibomian glands. It is more commonly seen in elderly people than in young subjects, especially if they are gouty. On everting the lids, numerous small, yellowish-white concretions will be seen distinctly gritty to the touch. These act like so many foreign bodies and produce considerable irritation and pain.

Each concretion should be removed with a fine needle, the conjunctiva having first been rendered insensitive with cocain.

Toxic conjunctivitis is a name suited to those forms of conjunctival inflammation caused by certain chemicals, by insects, and by the prolonged use of the mydriatics (notably atropin) and the miotics.

Atropin conjunctivitis occurs at all ages, but is commonest in old persons. Sometimes it will appear after only a few drops of the solution have been used, but usually not until the drug has been employed for a long time. It has been attributed to impurities in the drug, to the existence of free acid, to the presence of a fungoid growth, and to idiosyncrasy. In a number of instances arthritic history has been obtained (Collins). The disease usually appears in the form of follicular granulations, sometimes associated with much swelling of the lid and eczema of the surrounding tissue. (See also page 214.)

Eserin, hyoscyamin, duboisin, and homatropin less commonly cause this affection, and the same disorder has been reported as the result of the prolonged use of cocain.

Conjunctivitis occurs among those who work in anilin dyes, and from chryosphanic acid, when this has been used as an ointment in skin affections, and may be caused by artificial fertilizers. *Chrysarobin conjunctivitis* (photophobia, lacrimation, and blepharospasm) may, according to Igersheimer, be associated with gray deposits in the cornea,

resembling superficial punctate keratitis. Podophyllin coming in contact with the eye may produce intense congestion, edema of the lids, and infiltration of the cornea (C. Chiari).

Conjunctivitis caused by caustics, acids, and other strong irritants is elsewhere considered. Workers with *x*-rays are subject to a severe and at times intractable form of conjunctivitis (see page 507). Conjunctivitis may follow the stings of flies and other insects, and has been described as due to the presence of larvæ in the conjunctival sac (*larval conjunctivitis*). Parasitic conjunctivitis due to one of the groups of the higher fungi has been reported by A. J. Smith, C. M. Hosmer, J. T. Carpenter, Jr., and W. C. Posey. Violent conjunctivitis may be caused by the venom of certain serpents, by eel-blood, and contact with ascarides. Some persons quickly acquire a sharp conjunctivitis if they come near horses, for example, in driving, or if they stroke the fur of a cat or work among certain flowers, notably the primrose, or if plant hairs gain entrance to the conjunctival sac.

The *treatment* in general demands the removal of the cause, and in atropin conjunctivitis applications of tannin and glycerin and of an alum crystal are useful. In some instances the author has found a 1 per cent. solution of creolin of service. A bland ointment for the irritated cutaneous surface and the ordinary antiseptic lotions are indicated.

Conjunctivitis Nodosa (*Ophthalmia Nodosa; Pseudotuberculosis of the Conjunctiva* [Wangenmann]).—This disease is caused by the irritation of caterpillar hairs which have lodged in the conjunctiva, cornea, or iris, and was first described in 1883 by Pagenstecher. In addition to conjunctival congestion and pericorneal injection, the disease is characterized by a number of grayish or yellowish semitransparent nodules, which are located in the conjunctiva and episclera, the most usual situation being the ocular conjunctiva between the lower border of the cornea and the fornix. In a case studied by the author and E. A. Shumway, 27 such nodules could be differentiated, those directly in the center of the collection being somewhat confluent and assuming a crescentic and circular appearance. The lesions strongly suggest tubercle of the conjunctiva; and, indeed, the disease is called pseudotuberculosis of the conjunctiva by some authors. The center of each nodule usually contains a caterpillar hair, and is surrounded by round cells, giant cells, and externally by spindle cells and a capsule. It is not definitely decided whether the irritation is a mechanical one, or whether it is due to some constituent of the hairs. Not only is the conjunctiva affected, but the hairs may penetrate the cornea, enter the iris, and there form the nodules which have been described. It is probable that they may even reach the choroid. The disease is generally caused by certain species of caterpillar, particularly Lasiocampa, or Bombyx (*B. rubi, B. pini*), Liparis (*L. monacha, L. dispar*), etc. In the case studied by the author and Dr. Shumway the hairs of the *Spilosoma virginica* were identified. This subject has recently been elaborately studied by Teuschlaender and by Parker in this country. The *treatment* should consist in excision

of the conjunctival nodules and the ordinary remedies for conjunctivitis. A somewhat similar disease, clinically resembling trachoma, has been described by Markus as the result of the implantation of plant hairs.

Xerophthalmos (*atrophy of the conjunctiva; xerosis*) is the name employed by systematic writers to describe a dry, lusterless, and shrunken appearance of the conjunctiva, and is recognized under two forms—*parenchymatous* and *epithelial.*

The former type results from cicatricial changes which involve the deep layers of the conjunctiva; the sulcus is obliterated, and the lids, in severe cases, are attached to the eyeball, while the cornea is opaque. The surface of the conjunctiva of the lids is smooth, dry, and almost leathery to the touch. Granular lids, diphtheritic conjunctivitis, pemphigus, and essential shrinking of the conjunctiva are the causes of the disorder.

Treatment is of no avail, but some comfort may ensue by instilling glycerin and water or by the local use of an emulsion of cod-liver oil.

In the *epithelial type* the exposed ocular conjunctiva becomes dry and has a lack-luster appearance; cheesy flakes form, and the membrane is greasy and thrown into folds. A short bacillus (xerosis bacillus) has been found in the secretion of these cases, but its pathogenic character is doubtful. This form of xerosis sometimes occurs in epidemics, associated with night-blindness, and is seen among people of poor nutrition—for instance, during prolonged fasts—or among those whose eyes have long been exposed to sunlight. It is also one of the symptoms of keratomalacia in infants. According to Stephenson, the disease is not rare. Night-blindness is not always present, but usually there are signs of torpor of the retina, with contraction of the visual fields and reversal of the red and green fields (see also page 506).

The *treatment* demands a nutritious diet, a soothing collyrium, dark glasses, and removal from the surroundings which have caused the difficulty.

Amyloid disease of the conjunctiva is a rare disorder, in which pale, yellowish masses appear chiefly in its palpebral portion. It has been supposed to arise from granular conjunctivitis, but, according to Raehlmann, the growths are independent of trachoma.

Extirpation is the proper mode of treatment. Their structure is analogous to lymphoid tumors in which a hyaline degeneration may be found, and which, in all probability, is an antecedent condition (Raehlmann, Kubli). The diagnosis can be made with certainty only by submitting the tissue to the iodin test. Herbert has described hyaline or, as he prefers to call it, *colloid* degeneration of the conjunctiva.

Conjunctivitis Petrificans.—In this rare disease, described by Leber in 1895, a number of irregular white, opaque spots appear in the conjunctiva, which are slightly elevated above the surface and covered by epithelium. The surrounding conjunctiva is reddened and somewhat inflamed, and any portion of it may be affected, and in advanced stages the disease may spread to the bulbar conjunctiva and the tissue of the lids. The disease may assume a recurring as well as a spreading

nature, and in any event is a chronic one, and may last for months or even years. Nothing is known of the etiology of the disease, the white spots consisting of deposits of lime associated with an organic base. All cases thus far reported have occurred in young females. Recently Sidler-Huguenin observed a case of this character in a hysteric girl who produced the lesions by putting lime into the conjunctival sac. It did not differ from the type ordinarily described.

Pterygium is a peculiar, fleshy growth, consisting of hypertrophy of the conjunctiva and subconjunctival tissue. One or both eyes may be affected. Its most usual situation is at the inner side of the eyeball, corresponding to the course of the internal rectus muscle; more rarely it develops at the outer, and very exceptionally at the upper or lower, part. When the fan-shaped expansion arises from the semilunar fold and caruncle, it converges as it approaches the cornea, the center of which it rarely passes.

Fig. 109.—Large pseudopterygium, the result of a lime-burn.

The growth is uncommon in young subjects, the average age, according to Fuchs, being about forty-eight, although it often develops at a much earlier period of life. According to Robert Thompson, of Australia, and John McReynolds, of Texas, in their regions the affection is not infrequently encountered in young subjects, Thompson's youngest patient being only fifteen years old. The theory advanced by Arlt, that ulceration at the margin of the cornea should be regarded as the primary cause of the affection, is no longer tenable. According to Fuchs, pterygium is a development of a pinguecula, and like it, save in exceptional cases, belongs to the so-called senile changes in the eye. As the pterygium develops, the characters of the pinguecula disappear. Exposure to dust, smoke, wind, and heat is the exciting cause, according to McReynolds, who has often seen pterygia both at the inner and the outer canthus. *Pseudopterygia* may result from blennorrhea, burns, or erosions of the corneal surface, the thickened conjunctiva becoming attached to the corneal lesion.

The *treatment* consists in excision, transplantation, strangulation by means of ligatures, or evulsion (see page 614).

Pinguecula is a small, yellowish elevation situated in the conjunctiva near the margin of the cornea, and usually at the inner side. It has the appearance of fatty tissue, but is a hyaline degeneration of the connective-tissue fibers of the subconjunctival tissue, and should be regarded, according to Fuchs, as the first stage in the development of a pterygium. It may be excised or destroyed with the actual cautery.

Abscess of the conjunctiva is a rare condition, in which a localized area of suppuration appears in the subconjunctival tissues. It may develop in children of greatly depressed nutrition, and is sometimes

the sequel of a wound. *Ulcers of the conjunctiva* are occasionally seen and may be severe enough to destroy the tarsus (Cailloud). Widmark and T. Harrison Butler have described a form of conjunctival disease characterized by congestion of the inferior tarsal conjunctiva and of this membrane in the lower epibulbar expansion. Part or all of the affected area stains with fluorescein. Slight stippling of the cornea may be present. Brow pain and occipital headache may precede the inflammation. Massage with a 1 per cent. ointment of yellow oxid of mercury, according to Butler, is the best application in the treatment of *Widmark's conjunctivitis.*

Ecchymosis of the Conjunctiva.—This is an extravasation of blood beneath the conjunctiva scleræ, the meshes of the connective tissue being filled with blood-clot, and occurs as the result of an injury, or from some violent, straining effort—*e. g.*, during a paroxysm of whooping-cough or a convulsive seizure. It may arise without obvious cause, especially in elderly persons, and has been seen in young girls at the time of the menstrual epoch. Its occurrence during severe conjunctival inflammations has been described. *Recurring subconjunctival hemorrhages* are important indications of chronic nephritis and arteriosclerosis. They also occur in diabetes. Ordinarily, subconjunctival hemorrhage will subside by absorption and requires no treatment.

Chemosis (*edema*) **of the conjunctiva** occurs where the connective-tissue layer is distended with serum, and is often associated with an inflammatory exudation. It is generally a symptom of some other disease—for example, acute conjunctivitis, choroiditis, iritis, sinusitis, or orbital cellulitis. *Angioneurotic edema* of the conjunctiva, with swelling and hyperemia, may appear without any apparent cause and with marked suddenness. In paralysis of the external straight muscles the overlying conjunctiva is often decidedly edematous, and may be an early symptom of this condition. Chemosis of the conjunctiva following the use of iodid of potassium has been reported by the author, and it may succeed a general outbreak of urticaria.

Treatment.—The swollen tissues may be incised and an astringent lotion prescribed.

Emphysema of the conjunctiva consists in a distention of the connective-tissue spaces with air, and occurs under the same conditions which occasion this accident when it involves the eyelids.

Lymphangiectasis of the conjunctiva is a development of small blisters in the conjunctiva, filled with semitransparent fluid, and usually gathered together in masses. These are situated superficially, and readily move with the conjunctiva over the subjacent tissue. An interference with the natural lymph flow and consequent distention of the lymph-spaces is the probable explanation of their appearance. The affection is said to be most frequent in children, but may occur at any age. Spontaneous disappearance is the common outcome, but, if need be, the small blisters may be incised.

Syphilis of the Conjunctiva.—Chancres may develop on the upper or lower culdesac, and even upon the ocular conjunctiva, as

primary affections, and not only as extensions from the lids. A few instances of soft chancre have been described.

As manifestations of general syphilis, ulcerated papular syphilids and gumma of the conjunctiva have been recorded. Mucous patches occasionally develop on the conjunctiva. Finally, there is a type of inflammation called *syphilitic conjunctivitis*, which appears as a stubborn catarrh, or in the form of granulations similar to trachoma follicles, in an anemic and rather colloid-looking conjunctiva. The disease is not amenable to local treatment, but disappears under antisyphilitic remedies. Conjunctival lesions in hereditary syphilis are uncommon; papular syphilids and gummas have been described.

Tumors and Cysts of the Conjunctiva.—As congenital forms, translucent cysts, angiomas, cavernous angiomas, lymph-angiomas, dermoid growths (see page 284), and pigment spots have been described. *Moles of the conjunctiva* are usually deeply pigmented (occasionally they have a gray or pinkish color); rarely they spread over the tissue without involving the deeper layer. Angiomas may be situated on the palpebral conjunctiva, the bulbar conjunctiva, the fornix, or the plica. Usually congenital in origin, they may arise in later life. A nevus may be the starting-point of a sarcoma. Pigment spots, after healing of variolous pustules, have been described. *Nævus pigmentosus* also occurs (Wintersteiner) and may give rise to sarcoma. Pigment patches on the ocular and palpebral conjunctiva have been observed—*melanosis of the conjunctiva* (Randolph). In a patient under the care of the author there was marked bilateral pigmentation in both conjunctival sacs, with patches of pigment in the semilunar folds and caruncles. He was one of a family with this peculiarity, which had existed in a number of generations.

Cysts of the conjunctiva may appear in the bulbar conjunctiva, in the palpebral conjunctiva, and in the fornix. Parsons describes the following varieties: Retention, lymphatic, traumatic, parasitic, and congenital cysts. This materially simplifies Cirincione's elaborate classification. Retention cysts, often seen in the region of the retrotarsal folds, are small, oval, clear bodies. They develop in new-formed glands as the result of inflammation, in the so-called Henle's glands, and more rarely in Krause's glands. They may also arise from the accessory lacrimal gland. Lymphatic cysts appear in the bulbar conjunctiva, and represent dilatations of lymphatic vessels (lymphangiectasis and lymphangioma (see page 232). Usually multiple, they occasionally appear as isolated, yellowish cysts. Traumatic cysts arise as the result of a conjunctival wound or injury, and sometimes are *implantation cysts*—that is, through the wound epithelium from the skin, cilia, etc., gains entrance, degenerates, and produces the cyst. Parasitic cysts are usually due to the presence of a cysticercus, and appear as large, yellowish vesicles. A white spot in the wall may indicate the situation of the embryo. Filariæ may also cause conjunctival cysts. To a growth situated near the corneal margin, semitranslucent in color, and often of congenital origin, Parinaud gave the name *dermo-epithelioma*.

For this title Oatman preferred the name "epithelial cystoma of the conjunctiva," as he believed it represented a transitional stage in the development of the epithelial cyst.

Among the benign tumors, dermoids (see page 284), lipoma, fibroma, osteoma, granuloma, adenoma, hemangioma, and papilloma have their habitat upon the conjunctiva. Lipomas and lipomatous dermoids are found (see Fig. 114) between the superior and the external rectus. Fibromas are either hard or soft, and appear in the form of polypi. Soft fibromas occur chiefly in the fornix or palpebral conjunctiva, and are often highly vascular. Hard fibromas are less common, and arise from the palpebral conjunctiva or caruncle. Bone formation usually occurs betwen the margin of the cornea and the commissure. Papillomas

Fig. 110.—Epibulbar lipoma (from a patient in the University Hospital.)

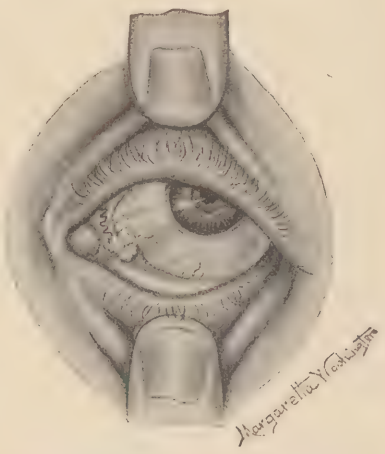


Fig. 111.—Papilloma of the conjunctiva (from a patient in the Philadelphia General Hospital).

are either pediculated or sessile, and histologically resemble the structure of the papillæ. They may arise from the conjunctiva or plica, and are often multiple. Ordinarily benign, they may undergo carcinomatous degeneration and infiltrate the eyelids (Risley and Shumway). Papillomas have been confounded with masses of granulation tissue arising from wounds—*e. g.*, after strabismus operations and with angiosarcomas. Adenomas may originate on the conjunctival surface of the lids from Krause's and Moll's glands, and from the Meibomian glands. They may develop into malignant growths—*adenocarcinomas.*

Treatment.—Usually the growths described can be readily excised, and the edges of the wound may be united with fine sutures. In simple cysts, cutting away the anterior wall is generally sufficient to cause a cure. Nevi have been treated with applications of ethylate of sodium

(Snell). The removal of papillomas should be thorough, as they have a tendency to undergo carcinomatous degeneration.

The malignant growths include epithelioma and sarcoma.

Epithelioma may occur as a primary growth upon the ocular conjunctiva, especially at the limbus corneæ, and rarely appears before the fortieth year of life. Commonly situated at the outer side, it may also appear on the nasal side, and in rare instances has surrounded the entire cornea (*peribulbar epithelioma*).

The epitheliomatous or carcinomatous growth usually first manifests itself as a small, reddish elevation surrounded by injection. Generally the growth is slow; its base is broad and attached to the underlying tissue; rarely a large fungous mass is formed. The tumor is composed of proliferating masses of epithelium which proceed from the

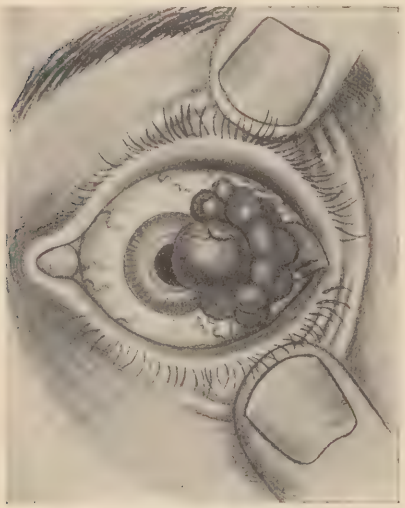


Fig. 112.—Sarcoma of corneoscleral junction (from a patient in the Jefferson College Hospital).

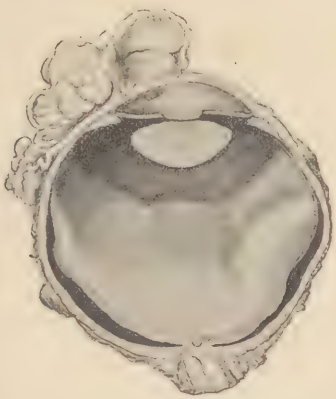


Fig. 113.—Section of eyeball (see Fig. 112) with sarcoma of corneoscleral junction.

surface epithelium, and are separated into alveoli by a connective-tissue stroma. As a rule, the substantia propria of the cornea is infiltrated, and if the growth involves the eyeball, it does so along the perivascular and perineural lymph-sheaths. Conjunctival epithelioma may be pigmented (*melanocarcinoma*).

Sarcoma of the conjunctiva arises at the limbus (*epibulbar sarcoma*), its subjects generally being past middle life, in the form of a reddish-white or brownish-black growth, usually overlapping the cornea, but not often involving its structure. Both pigmented and unpigmented varieties occur, the former being the more frequent. The tumors may grow rapidly and reach a large size. They are composed of round and spindle cells, and may have a markedly alveolar arrangement (*alveolar sarcoma*); sometimes the cells are distinctly epithelioid in type (*endo-*

thelioma). Epibulbar sarcomas may develop from collections of pigmented cells on the conjunctiva (*pigment spots, melanomas*). Rarely they invade the interior of the eye. Sometimes they are multiple. Sarcomas of the palpebral conjunctiva and fornix and diffuse melanotic sarcomas have been recorded. *Angiosarcomas of the conjunctiva* arise from a proliferation of the adventitia of the blood-vessels, and in their growth, like fibromas, they thrust the epithelial covering in front of them. They have been mistaken for papillomas.

Prognosis and Treatment.—Epitheliomas vary in malignancy; but they tend to recur even when superficial. Occasionally it may be proper to remove the growth, with the expectation of saving the eyeball; but if it is involved, complete removal of the globe is indicated. Epibulbar sarcomas have been removed and the eyeball preserved; but Verhoeff and Loring regard them as highly malignant, inasmuch as there is a history of recurrence in fully 80 per cent. of the cases, and recommend that the eyeball should be removed at once. Excision of

Fig. 114.—Lipomatous dermoid of the conjunctiva: eye turned up and in.

Fig. 115.—Sarcoma of the conjunctiva (from a patient in the Philadelphia General Hospital).

these growths is sometimes practicable in the early stages without sacrificing the eyeball.

Lepra.—According to Lopez, the chief alterations in the conjunctiva produced by leprosy are anesthesia, inflammation, pterygia, and tubercles. The anesthesia of the conjunctiva probably determines the chronic conjunctivitis, which is common. Pterygia are frequently observed, and are caused by the action of external irritants upon the ocular conjunctiva, which has become insensitive under the influence of the disease.

It is convenient in this place to refer to the effect of *leprosy upon the cornea*, in which the lesions are frequent and varied. The tubercles which form in the conjunctiva are apt to attack the corneoscleral margin, but may involve the cornea exclusively. A late manifestation of the disease is an inflammation of the cornea known as *leprous keratitis*, which somewhat resembles interstitial keratitis.

Lupus occurs as a primary disease, or extends to the conjunctiva

from the surrounding integument. It appears in the form of red, granular patches placed upon an ulcerated base. As the same microbe is the cause of lupus and tuberculosis, any difference existing in the two diseases occurring in this situation must rest upon the clinical appearances, the lupus spot showing healing in one direction and active ulceration in another. Those cases in which the disease has spread from the lid to the conjunctiva have especially been classified as lupus.

Tubercle of the conjunctiva occurs as a *primary* and as a *secondary* affection.

Primary tuberculosis of the conjunctiva is rare, but a certain number of instances are upon record in which there was an absence of evidence of tuberculosis elsewhere, and in which there was no reappearance of the disease locally, or in distant organs, after its removal. Villard maintains that in 60 per cent. of the cases he has analyzed no initial tuberculous lesion was discovered, and therefore he does not believe in its endogenous origin.

As a secondary affection it has usually appeared in association with nasal and laryngeal tuberculosis.

According to Eyre, who adopts Sattler's classification, the disease may appear in one or other of the following manifestations: (1) One or more miliary ulcers which usually caseate; (2) grayish or yellowish subconjunctival nodules which resemble the sago granules of trachoma; (3) florid hypertrophied papillæ and rounded, flattened outgrowths of granulation tissue; (4) numerous pedunculated cock's-comb excrescences; (5) a distinctly pediculated tumor. The ulcers have uneven and slightly raised edges, and their floors have yellow or sometimes a lardaceous appearance.

There are thickening of the lids, dark-red swelling of the conjunctiva, considerable discharge, and occasionally tumefaction of the tear-sac. The preauricular and submaxillary lymphatic glands of the same side are enlarged. Pain is not considerable unless the ulceration involves the bulbar conjunctiva and cornea or extends to the lids.

The disease should be distinguished from trachoma, epithelioma, and syphilitic ulceration.

Diagnosis.—In any suspected case the real nature of the affection may be decided by excising a portion of the diseased tissue, submitting it to microscopic and bacteriologic examination, or by submitting the patient to a test with tuberculin, or by implanting a portion of it in the anterior chamber of a rabbit's eye. It is not always possible to demonstrate the presence of tubercle bacilli.

In trachoma the lymph-glands are not involved and the discovery of the Prowazek bodies (see Fig. 105) would tend to establish the diagnosis.

Epithelioma is excluded by the age of the subjects, tuberculosis almost invariably occurring in young persons; that is, those under the thirtieth year of life.

Prognosis.—This depends upon whether the disease is primary or secondary. In order to prevent general infection it is important to

eradicate the local lesion. Sight may be destroyed by involvement of the cornea.

Treatment.—The diseased tissue may be removed with a knife or curet; the galvanocautery has been recommended. The subsequent treatment should include the use of a collyrium of bichlorid of mercury, and iodoform or aristol powder. Injections of *tuberculin TR* are advisable, and, according to Ormond and Eyre, represent a treatment far superior to incision and scraping. Stephenson suggests the trial of the *x*-rays. Axenfeld uses a 50 per cent. solution of lactic acid in the treatment of conjunctival tuberculosis; after its application the eye is irrigated with a saline solution.

Pemphigus of the conjunctiva is a rare affection, characterized by the formation of bullæ, associated with pain and lacrimation, and, after succeeding attacks, degeneration and cicatrization of the conjunctiva. Instead of vesicles on the inflamed area, membranous exudations, grayish-white in color, may form. According to Michel, the disease may be confined to the conjunctiva, or it may attack not only the conjunctiva, but also the mucous membrane of the nose, mouth and pharynx, and the skin.

The course of the disease, which tends to recur from time to time, is destructive to the nutrition of the conjunctiva, and later to the cornea. The former undergoes cicatricial change, and may grow fast to the ball; the latter becomes opaque and staphylomatous.

Under the name *essential shrinking of the conjunctiva*, a condition of atrophy, contraction, and gradual disappearance of the conjunctival culdesac has been described, during which the free borders of the lids become fixed to the ball and the cornea becomes dry and opaque. This probably is a form of pemphigus, but has also been recorded as an essentially distinct process. According to Pergens, essential shrinking of the conjunctiva may be produced by trachoma, psoriasis, xeroderma pigmentosum, ichthyosis, and lupus.

Treatment.—This is practically unavailing. Applications of glycerin and water and other emollients have been employed with the hope of keeping the conjunctiva moist, and *x*-rays (Neeper) and thiosinamin (Melville Black) have been advised. Rabbits' conjunctiva and human conjunctiva have been transplanted, but usually without beneficial results. The internal administration of arsenic has been recommended.

Injuries of the Conjunctiva.—(*a*) **Foreign Bodies.**—A small particle of coal, ash, or dust is easily removed if lodged upon the lower portion of the conjunctiva; but if it finds its way beneath the upper lid, and is situated far back under the retrotarsal fold, it may not come into view when the lid is everted unless the fold is pushed into prominence. If the foreign body is attached to the tissues it may be necessary to dislodge it with the point of a needle or with a spud. Cocain or holocain will render this operation painless.

(*b*) **Wounds.**—These may be part of a serious injury involving the lid or deeper structures of the eye; more rarely they occur as simple lacerations, confined usually to the bulbar portion. In suitable cases,

after proper cleansing, the lips of the wound should be drawn together with a few sutures.

(*c*) **Burns.**—These are commonly inflicted with lime (mortar or quicklime), molten metals, powder, and acids, and are especially serious because of the deformity which the subsequent contraction is likely to produce, or on account of the development of a symblepharon (see page 180). Ulceration of the cornea, hypopyon, and even panophthalmitis may result. The prognosis of such injuries is always grave.

All foreign substances must be removed at once, and if lime has been the injuring agent, this is best accomplished by forcible irrigation of the conjunctival sac with clean water. Schmidt-Rimpler, however, prefers, in these circumstances, thorough cleansing of the eye with oil introduced with a syringe into the culdesac. For acid burns an alkaline lotion is usually recommended. The subsequent treatment consists in the instillation of olive or castor oil, and atropin drops, to prevent secondary iritis if the cornea is much inflamed; atropin may be incorporated with liquid vaselin and placed in the culdesac. Powder grains are frequently embedded in the conjunctiva; they cannot be picked from their beds; usually the larger ones can be excised.

Affections of the Caruncle.—The caruncle and semilunar fold may be swollen in conjunction with a general inflammation of the conjunctiva, but also may undergo localized enlargement and inflammation, to which the name *encanthis* has been applied, and which is subdivided by systematic writers into an *acute* or *inflammatory* and a *chronic* variety. The process may go on to the formation of a minute abscess.

Swollen caruncles are commonly found in patients with eye-strain, especially with imperfect amplitude of convergence. The small body is red, elevated, and angry looking, and injected vessels run from it toward the cornea in the interpalpebral space. This condition might be designated *symptomatic* or *functional encanthis.*

In like manner, temporary irritation of the structure is caused by the lodgment upon it of a foreign body, or by the presence of misplaced cilia which rub against it. The caruncle should be carefully examined when patients complain of irritation, lacrimation, and inability to use their eyes with comfort.

The excessive development of the hairs normally placed upon the caruncle is called *trichosis caruncul*æ.

A number of tumors situated upon and growing from the caruncle have been recorded; in two instances the growth proved to be an adenoma (Prudden and Schirmer). Primary sarcoma (Veasey, Snell) and carcinoma of the caruncle (*malignant encanthis*) have been described. Papilloma, dermoids, nevus, fibroma, lymphangioma, epithelioma, cylindroma, angiosarcoma, and lymphosarcoma have also been reported (V. Berl).

Treatment.—Local irritations of this body may be relieved by the direct application of a mild astringent like alum, or soothed by touching it with tincture of opium. Foreign bodies, stiff hairs, and misplaced cilia must be extracted. A tumor should be removed by excision.

Argyria Conjunctivæ (*Argyrosis*).—Long-continued application of solutions of nitrate of silver to the conjunctiva may be followed by a brownish discoloration of this membrane. For this reason it is inadvisable to allow patients to use at home even a weak collyrium of this drug. The same discoloration follows the injudicious use of protargol, argyrol, and largin; indeed, these drugs produce the stain more quickly than nitrate of silver, even, it is said, after a few weeks of their employment. The coloration is due to staining of the elastic fibers; the epithelium is free from pigment. Argyrosis from nitrate of silver is practically irremediable, although the use of dionin is said to decrease its intensity (Lebensohn). Argyrosis from argyrol decreases after a discontinuance of the drug (Krauss). A collyrium of iodid of potassium (0.5 per cent.) may be tried. A yellowish-brown discoloration of the conjunctiva, known as *siderosis conjunctivæ*, due to the prolonged use of sulphate of iron, has been reported.

CHAPTER VII

DISEASES OF THE CORNEA

Under the general term *keratitis* are included divers forms of inflammatory affections of the cornea, to which, according to the type, certain well-marked stages belong; cellular *infiltration* in the layers of the cornea going on either to absorption or to the formation of pus; loss of the substance of the cornea lying over the infiltrated area, and the development of an *ulcer;* loss of the transparency of the superficial corneal layers over an infiltrated area, which has been converted into pus and created an *abscess,* with the final destruction of these layers by the development of the abscess; the appearance of *vessels in the cornea;* and the process of *repair* after loss of substance, or the period of *cicatrization.*

Certain associated and subjective symptoms may be present in all forms of corneal inflammation. Among the former the most notable are the congestion of the vessels of the circumcorneal area; involvement of the iris and ciliary body in the severe types of the affection, with the added signs of iritis and the development of exudation in the anterior chamber. The subjective symptoms include diminution of vision, pain, photophobia, excessive lacrimation, and blepharospasm.

Although it is customary to divide the many types of corneal inflammation into suitable groups, it is by no means possible to refer the disease in each instance to one or other of these divisions.

Phlyctenular Keratitis or Keratoconjunctivitis (*Eczema of the Cornea*).—This disease is characterized by the formation of single or multiple phlyctenules on some portion of the cornea, and is accompanied by dread of light, excessive lacrimation, and blepharospasm.

Causes.—The disease is commonly seen in so-called scrofulous subjects, rarely before the first year of life, most frequently in children before the age of puberty, and less commonly in adults. It often is secondary to phlyctenular conjunctivitis or is associated with it (see page 211). Enlarged lymphatic glands, prominent and swollen lips, and diseases of the joints and bones may be present.

This form of keratitis is in close connection with obstructive (adenoid vegetations) and inflammatory diseases of the nasal passages, and an infectious rhinitis is a constantly associated disorder, which, in turn, determines an eczema about the nares. Indeed, the clinical association between this disease and eczema is intimate, and eczema of the face and scalp is frequently an accompanying condition. The affection often follows in the wake of measles or other acute exanthemata, and is distinctly under the influence of climate, being aggravated in warm and moist weather. Tuberculosis of the lymphatic glands is present in

fully one-half of its subjects, and the evidence is daily increasing that phlyctenular keratitis is closely connected with tuberculosis and probably caused by it. A large number of the subjects of phlyctenular disease (88 per cent., G. S. Derby, Stock) react to tuberculin—a suggestive fact, although it does not prove that tuberculosis is the cause of the disease. Phlyctenules not infrequently have developed as the result of the Calmette reaction and have also followed subcutaneous injections of tuberculin.

Staphylococcus pyogenes aureus and *albus* are present in the epithelium of the affected regions; but these organisms are usually not found in any number in fresh phlyctenules. Tubercle bacilli have not been discovered (L. Müller). The exact cause of the ocular lesions, or phlyctenular eruption, has not been determined.

Symptoms.—The phlyctenules, which consist in the early stage of minute subepithelial collections of round cells, appear upon the cornea usually at or near the corneoscleral junction. They vary in size from a poppy-seed to a millet-seed; their tops, at first gray, speedily grow yellow, break down, and form superficial ulcers. They are accompanied by decided local congestion, increased lacrimation, and photophobia.

The palpebral conjunctiva, always hyperemic, may remain translucent and bathed in tears, or the disorder may be complicated with mucopurulent conjunctivitis.

When the photophobia is severe, the child buries its head deeply in the bed-clothes; the lids are spasmodically closed, rendering inspection of the eye difficult, at times well-nigh impossible. The dread of light and the blepharospasm are probably due to direct irritation of the corneal nerves, as Iwanoff found the cellular infiltration situated along their course.

The pustule, when it breaks down, forms a *phlyctenular ulcer*.

This may remain at its original seat near the margin, or creep toward the center of the cornea (*migratory pustule*), followed by a bundle of thickly crowded blood-vessels, and form a special type of corneal inflammation, known as *fascicular keratitis*. The blood-vessels, when the ulcer heals, disappear, but a stripe of opacity remains.

Under the name *phlyctenular marginal keratitis* a variety of this disorder exists, characterized by the development of numerous phlyctenules along the rim of the cornea, giving rise to a process which may cease here, or which, by further invasions, may produce vascular ulcers.

More dangerous than any of the other varieties is the formation of a *single pustule*, just at the corneal border, which speedily ulcerates and is surrounded by a yellow area of infiltration, with a strong tendency to perforate.

If these inflammations recur constantly, the cornea becomes clouded, uneven from loss of epithelium, and covered with numerous superficial vessels, the whole forming the so-called *phlyctenular pannus*.

Sometimes in the middle and deep layers of the cornea extensive

gray or yellow opacities may form, which may suppurate with large loss of tissue, or go on to resorption. These are the so-called *deep scrofulous infiltrations.*

Pathology.—The efflorescence or phlyctenule consists of a collection of lymphoid cells, lying between Bowman's membrane and the epithelium, by the softening of which, as before described, the superficial cells are discharged, and an open, ulcerating surface is exposed. By further degeneration the entire nodule disappears, and the loss of substance is rapidly replaced with epithelium.

Diagnosis.—This presents no difficulties, direct inspection rendering the nature of the disease evident.

Prognosis.—The course varies greatly; in mild cases healing takes place with only a slight loss of substance, and the resulting scar is scarcely discernible.

Not so with the severe forms, in which there has been decided loss of substance and a distinct scar-tissue remains, or in which deep ulceration with perforation occurs, or where constantly recurring vascular ulceration leaves an uneven and roughened surface. In children of the tuberculous type, especially if their surroundings are unfavorable, phlyctenular keratitis is exceedingly intractable.

Treatment.—In order to make a thorough application of the local remedies the child's head should be taken between the surgeon's knees and the lids separated, while the attendant holds the hands and body; the cornea will usually roll out of sight, but gradually may be coaxed into view. Sometimes a lid-elevator is useful, and a few whiffs of ether or chloroform may be necessary.

If much secretion is present, boric acid or physiologic salt solution should be employed, and atropin drops should be instilled with sufficient frequency to maintain mydriasis. Cocain, judiciously used, will allay the photophobia, but its continuous application where corneal ulcers exist is to be deprecated. Holocain is sometimes useful; dionin occasionally seems to act unfavorably in phlyctenular keratitis. An ointment of the yellow oxid of mercury, gr. j to 3j (0.065–3.885 gm.), either with or without the addition of atropin, may be employed, or calomel be dusted into the conjunctival sac, provided no form of iodin is being exhibited (see page 212). The eyes should be protected with goggles, and the child encouraged not to bury its head in the bedclothes (see also page 251).

Douching the eyes with cold water will subdue the dread of light, and touching the ulcerated external commissure, which almost invariably exists in these cases, with a crystal of bluestone, as Koller has suggested, helps to relieve the blepharospasm. In severe cases the ulcerated fissure may be incised, or the lids may be forcibly separated. No doubt this acts by stretching or rupturing a few fibers at the commissural angle, and relieves the spasm in the same manner as a similar manipulation is efficacious in fissure of the anus.

The best possible hygienic surroundings must be obtained, with fresh air and wholesome food. Cod-liver oil, iron, especially syrup of the

iodid of iron, syrup of hydriodic acid, quinin, often suitably given with pepsin, and arsenic are the most acceptable internal remedies.

The urine should be examined in all these cases; and scrupulous attention to the condition of the alimentary canal is an important factor in the treatment.

If rhinitis is present, a powder composed of equal parts of pulverized camphor, boric acid, and subnitrate of bismuth is useful (Augagneur), especially if the parts are thoroughly cleansed with Dobell's solution before its insufflation into the nasal chambers; powdered iodoform may be used in like manner, but its odor is objectionable, hence nosophen is preferred by many surgeons, and borobismuth ointment is recommended. The affected mucous membrane may be painted with compound tincture of benzoin or sprayed with permanganate of potassium (1 : 5000). In obstructive postpharyngeal and nasal affections (hypertrophies, adenoid vegetations) the diseased areas must be treated according to the methods of intranasal surgery. A patulous condition of the lacrimal passages should be secured.

In stubborn forms of recurring vascular ulcer and deep ulceration, especially in the fascicular type, the use of the actual cautery in the manner later described is productive of excellent results, or the ulcer may be touched with trichloracetic acid. In general terms, the treatment of severe types of phlyctenular ulcer is the same as that recorded on pages 252–254. In phlyctenular pannus *peritomy* is sometimes a useful procedure, and *canthoplasty* may be necessary.

After healing, provided the condition of the cornea permits it, any refractive error should be corrected. The best results in the treatment of phlyctenular disease are secured if its subjects are treated like other cases of tuberculosis—viz., after the patient is instructed as to general living, proper food, etc., he is visited in his home by one of the class-workers in the social service of the hospital, and is shown how to live and helped to carry out all directions. This method, advocated by George Derby, the author has followed with satisfaction in his hospital patients.

In general terms, phlyctenular inflammation of the cornea, which has just been described, is a circumscribed, usually superficial keratitis, and is known under a variety of synonyms—lymphatic, scrofulous, vesicular, fascicular, and pustular—and where it appears in adults assumes the form of a simple corneal infiltration. It furnishes the greatest number of ulcers of the cornea which are found in early life, and also a large group of those ulcers which are of *primary* origin—*i. e.*, where the disease starts in the cornea, the remainder of the group being caused by injury, abscess, depressed nutrition, etc. The entire series is in contrast to *secondary* ulcers—*i. e.*, where the disease follows as the result of a severe inflammation of the conjunctiva—*e. g.*, purulent, diphtheritic, or granular conjunctivitis.

The remaining inflammations of the cornea are divided by systematic writers into *ulcerative* and *non-ulcerative* inflammations.

Ulcers of the Cornea.—If the stage of infiltration fails to termi-

nate in absorption and there is destruction of the overlying corneal tissue, an ulcer results. Surrounding the area of necrotic tissue is a clear space, and beyond this a ring of infiltrating leukocytes which come from the vessels at the edge of the cornea. In favorable cases this necrotic tissue is thrown off, the surrounding cornea clears, the ulcer is covered by a proliferation of the epithelium, and the loss of substance is replaced by connective tissue derived from the fixed cells of the cornea. Where the process is progressive, successive layers of the cornea become involved, the iris and ciliary bodies are infiltrated, and hypopyon forms (see page 247). If the ulceration is not checked, the cornea perforates, and inclusion of the iris may result in *staphyloma* (see page 259). If the iris does not prolapse, the perforation may be closed with a tissue pro-

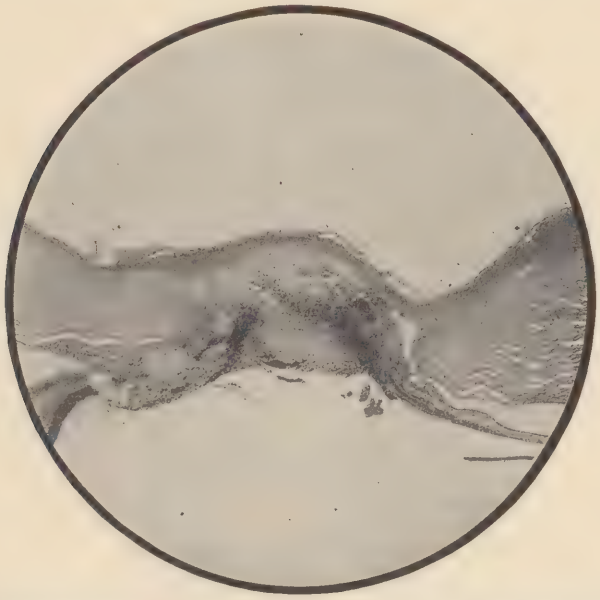


Fig. 116.—Perforating ulcer of the cornea with incarceration of iris (from a photomicrograph).

duced by proliferation of the posterior lining endothelium. Bowman's and Descemet's membranes are never reproduced.

In addition to those which have been described with phlyctenular keratitis, corneal ulcers may be gathered into several groups:

1. **Simple ulcer** appears in the form of a small, superficial, gray lesion, associated with slight pericorneal vascularity, and results from the rupture of a phlyctenule ("pimple ulcer") or from trauma.

An ulcer, which, from its situation, is called *small central ulcer*, may develop as a gray or gray-white opacity in the center of the cornea, and is not accompanied with much vascularity or dread of light. The elevation is slightly cone shaped until the whitish top breaks down into a shallow depression.

Usually single, this form of ulcer may be multiple, and tends to recur. It is seen in young chilren who have been poorly nourished and are of a so-called strumous habit. While healing generally occurs with promptness, a permanent opacity may remain, which, from its central situation, may seriously impair vision. If neglected, and in patients of poor nutrition, this ulcer occasionally forms an abscess of the cornea, or changes its type and develops into the following variety:

2. **Purulent or deep ulcer** consists of an area of yellowish (purulent) infiltration, surrounded by a zone of hazy cornea, round or irregular in shape, centrally excavated, and with a tendency to travel inward toward perforation, but not to extend in a lateral direction. Like all severe types of corneal ulceration, it may be associated with inflammation of the iris and the formation of pus in the anterior chamber; if perforation takes place, an adherent scar or leukoma results.

This ulcer is either *primary* from injury, and sometimes contains a foreign body as its nucleus, or is *secondary* to a violent grade of conjunctival inflammation. The subjective symptoms are pain, brow-ache, congestion, and sometimes, though not necessarily, photophobia.

3. **Indolent ulcer** (*absorption ulcer*) occurs under several forms: (*a*) *Shallow central ulcer*, with slightly turbid base, unattended with any considerable pain or photophobia, essentially chronic in its course, and healing finally with a faintly opaque facet (*faceted ulcer*).

(*b*) *Excavated* or *gouged-out ulcer*, often seen in children, most troublesome because it is rebellious to treatment, has its seat near the corneal margin. It may be entirely overlooked on account of the absence of congestion, and because in appearance it is a small, punched-out excavation with transparent bottom, and free from any opaque surrounding. The floor of the ulcer loses its translucency when healing is about to take place, and a few vessels of repair pass to its margin.

(*c*) *Reparative ulcers* are seen when, as occasionally occurs in the course of the healing of an ordinary corneal ulcer, this loses its turbidity and assumes a clear, facet-like appearance. These are similar to the absorption ulcers which occur primarily, and which, unattended with injection and with local symptoms, may, none the less, extend inward and perforate the cornea.

Indolent ulcers, in general terms, depend upon some failure in the nutrition of the cornea due to nervous disturbance. They are found in anemic and scrofulous subjects, and are seen in cases of chronic trachoma.

4. **Infected or Sloughing Ulcer** (*Purulent Keratitis*).—Ulcers without vessels of repair, which spread widely from one border and readily become complicated with hypopyon and iritis, and which are often the result of a trifling injury, usually affect elderly persons and those whose nutrition is depressed.

The most important type of these is the *acute serpiginous* or *creeping ulcer* of Saemisch. In the beginning a nearly central gray area forms, which ulcerates; its margins are sharp, and one, assuming the form of an elevated curve, is more decidedly opaque or yellow than the others, and

is known as the *arc of propagation*. Immediately behind it, the ulcer with its gray floor seems deeper than the portion next to the corneal margin.

The surrounding cornea is opaque, and the lesion spreads rapidly, at the same time growing deeper; iritis, iridocyclitis, and hypopyon ensue, and perforation and extensive sloughing of the cornea are likely to occur. Usually the patient complains of severe brow-pain and the eye is intensely tender. Vision is reduced to mere light perception. In other cases, while the local lesion is severe, the subjective symptoms of inflammation are almost absent. Kipp calls attention to certain types of infected ulcer from the margin of which straight, or nearly straight, lines diverge in all directions obliquely through the parenchyma of the deepest layers, their ends being connected by intermediate striæ. They may be due to folds in Descemet's membrane or to cell infiltration. Their presence indicates a favorable prognosis.

Hypopyon, to which reference has just been made, may be seen with both small and large ulcers, and consists of a collection of pus in the anterior chamber, varying in extent from a mere line to a quantity which well-nigh fills the chamber.

This appears as a yellow mass at the bottom of the anterior chamber, and is bounded above by a horizontal margin. If the collection is fluid, its position will shift with movements of the head; if it is tenacious, no movement can be observed. The pus is caused by an aggregation of leukocytes derived from the vessels about the periphery of the cornea and from those in the inflamed ciliary body and iris, the endothelium of which is cast off. In other words, the pus in hypopyon does not come from the cornea. Sometimes Descemet's membrane is ruptured without perforation of the cornea, and the pus in the cornea and in the anterior chamber is in direct connection.

Fig. 117.—Infected ulcer of the cornea, with hypopyon—hypopyon keratitis (modified from Haab).

The combination of ulcer of the cornea and pus in the anterior chamber has received the name *hypopyon keratitis*, which generally is limited to the type described as infective or creeping ulcer.

Causes of Infected or Sloughing Ulcers.—The investigations of Uhthoff and Axenfeld have demonstrated that—(1) Typical serpiginous ulcer of the cornea with hypopyon is nearly always caused by the *pneumococcus* (Fränkel-Weichselbaum capsulated diplococcus); this microorganism may frequently be found in these ulcers in almost pure cultures. These ulcers are also occasionally caused by the diplobacillus of Morax and Axenfeld (*diplobacillary ulcers*), the bacillus of Petit, the Bacillus subtilis (Zur Nedden), and the streptococcus. (2) Sloughing

ulcers not typically serpiginous are caused by infection with *staphylococci, streptococci*, and by *mixed infection*. Occasionally pneumococci originate ulcers which are not characteristically creeping. (3) In addition to the micro-organisms mentioned, the following bacteria, according to Uhthoff, have been found to be the cause of various forms of infected ulcer: Pfeiffer's capsulated bacillus, Bacillus pyogenes fœtidus, Bacterium coli, Bacillus pyocyaneus, diplobacillus, pneumobacillus, ozena bacillus, tubercle, and lepra bacillus. Other unidentified varieties have also been found, and corneal ulceration has also been ascribed to *streptothrix* (De Bernardinis).

Infected ulcer due to *Bacillus pyocyaneus*, as a rule, is a malignant process. Preceded by superficial keratitis a deep lesion develops, without the crescentic shape of the serpiginous ulcer and containing pus, chiefly in its center. The diagnosis depends upon a bacteriologic examination.

5. **Mycotic Keratitis** (*Keratomycosis Aspergillina*).—In a small percentage of cases of sloughing keratitis the infection is due to a mold—*Aspergillus fumigatus*—the fungus, as a rule, finding entrance through a corneal abrasion from injury. The ulcer has a dry appearance and has been compared to a grease-spot; it is surrounded by a gray or yellow line, and the enclosed area ultimately exfoliates. Hypopyon and iritis may be present; sometimes the lesions assume the form of a simple corneal infiltration resembling fascicular keratitis. Ellett has reported a corneal ulcer in which he found *Aspergillus nigricans*. According to Morax, mycotic corneal affections may be due to the *Verticillium graphii*.

The various micro-organisms come from the conjunctiva, the ciliary borders, the nares, the lacrimal passages, and from external contaminated surroundings. An injury to the cornea from a chip of stone, a chestnut-bur, beard of wheat, or the like may become infected, and be the starting-point of these dangerous forms of corneal ulceration. Typical serpent ulcer is rare in children, whose corneas appear to withstand pneumococcal infection.

6. **Abscess of the cornea** consists of a purulent infiltration in the deeper layers of this membrane, over the center of which, in the early stages, the epithelium is unbroken and prominent, but later, discolored and slightly sunken.

The corneal zone immediately surrounding it is hazy. The margins of the collection are thicker and more prominent than its middle; pus is seen in the anterior chamber; the aqueous humor is turbid and the iris inflamed.

Generally the lesion grows more yellow, notches laterally, bulges forward, and finally bursts, leaving a more or less ragged ulcer covered with tenacious pus, and pursuing a course similar to or identical with that described under sloughing or infected ulcer, of which, indeed, abscess is the first stage. The causes are identical with those described in connection with infected ulcer; occasionally a definite cause cannot be ascertained.

7. **Infected Marginal Ulcer.**—According to Zur Nedden, this form of corneal disease consists of a 1 to 2 mm. long oval ulcer, running parallel to the limbus, with only a slight diffuse infiltration in its vicinity, the rest of the cornea being normal. Sometimes several superficial infiltrations develop, which may unite with the original ulcer and form a sickle-shaped lesion; exceptionally the cornea is completely surrounded. In other cases the multiple infiltrations do not coalesce. The infection is believed to be due to a specific micro-organism, to which Zur Nedden gives the name "bacillus of infected marginal ulcer." The prognosis is good; hypopyon rarely forms.

8. **Exanthematous Keratitis.**—Most violent forms of *suppurative keratitis* occur during the convalescent stages of small-pox, though pustules rarely form upon the cornea. Abscess of the cornea occasionally accompanies scarlet fever, influenza, measles, typhoid fever, typhus fever, and pyemia, and in these cases has been regarded as metastatic, the pathogenic material having been conveyed through the blood, and not as coming from without, as in the more usual examples. Schirmer's investigations indicate, however, that the so-called *variolar abscess of the cornea*, which has hitherto been considered an endogenous infection, arises by penetration of the virus from without.

Fig. 118.—Mooren's ulcer (from a patient in the University Hospital).

9. **Ulcus rodens** is the name which was applied by Mooren to a *creeping ulcer* which begins at the upper edge of the cornea as a superficial lesion, separated from the healthy portion by a gray, opaque rim, which is undermined. The deeply undermined conjunctival edge of the ulcer is a striking feature of the disease. The extent of the undermining may reach 4 to 5 mm. from the border of the cornea (Randolph). Mooren's ulcer may be associated with an ulcer of the sclera (Parsons). Although vessels may pass to a rodent ulcer and cicatrization apparently begin, it relapses quickly and progresses forward, until the whole cornea has been traversed and sight is destroyed. The cornea is not usually perforated in this disease, which is a rare form, sometimes bilateral, attacking adult and depressed subjects. The process may last from two to ten months and even longer. It is called by Nettleship *chronic serpiginous ulcer* and *Mooren's ulcer*. The cause of the disease has not been discovered.

Fuchs has described *keratitis marginalis superficialis*, that is, a superficial variety of ulceration, which he has encountered in middle-aged

persons, and which proceeds somewhat unevenly from the corneal border, so that the margin is indented toward the corneal center and framed in a gray line. The conjunctiva may be attached to it in the form of a pseudopterygium. The lesion differs from ulcus rodens because it is more superficial and less undermined. It may last for long periods of time and is subject to relapse.

10. **Circular ulcer** (*marginal ring ulcer, annular ulcer*) occurs in the form of a deep groove at the corneal margin, which gradually progresses until it may entirely girdle the cornea and cut it off from its nutrition. Photophobia, injection, lacrimation, and other irritative symptoms are not prominent, but perforation of the cornea and prolapse of the iris are common. The disease is seen in debilitated subjects.

Another variety of *ring ulcer* is formed as the result of a *marginal phlyctenular keratitis* (see page 242), probably by the coalescence of a number of small foci. Ring ulcers are also seen in catarrhal and purulent conjunctivitis, and in the latter condition may prove especially dangerous if they are hidden by the chemotic conjunctiva.

11. **Dendriform ulcers** (*keratitis dendritica; ulcerans mycotica; furrow-keratitis; kératite ulcéreuse en sillons étoiles*) are forms of keratitis which appear in branch-like ramifications, having a superficial situation, with slight knob-like swellings at the end of the branches. The cornea may be insensitive and fluorescein will stain not only the lesions, but the cornea exclusive of them (Verhoeff). The inflammation manifests itself in two forms.

In one, from the beginning, the symptoms include photophobia, lacrimation, strong bulbar injection, swelling of the upper lids, and absence of the epithelium over the furrow-formed ramifications—an implantation of the process in the deeper corneal layers.

In the other the disease assumes a subacute or torpid character, with practical absence of severe irritative symptoms and loss of the covering epithelium—a limitation of the lesion to the superficial layer. In the first form the opacity is confined to the axis of the furrows; in the second, to the border. After healing, the scars have the same general configuration which was present during the stage of ulceration. The disease occurs in both sexes, and occasionally is seen in children.

The *cause* is not definitely known. The disease is attributed by C. J. Charles to a terminal nerve lesion, and by Verhoeff, who classified it with neuropathic affections of the cornea, to disturbance of the nerve-supply. Fuchs thinks some cases may arise from febrile herpes of the cornea (see page 264) by the increase and coalescence of the small blebs.

A keratitis in which the lesion consists of a peculiar, narrow, serpiginous, superficial ulcer, with lateral offshoots, like the skeleton of veins in a lanceolate leaf, usually accompanied with photophobia and lacrimation, and sometimes ushered in with severe supra-orbital neuralgia, has been attributed to malaria. It is a form of dendritic keratitis and has been well studied by Kipp and by Ellett.

12. **Exhaustion ulcer** (*keratomalacia*) may appear as an extensive ulceration in the center of the cornea, or as a ring abscess at its circum-

ference. The tissue speedily is converted into a slough, which drops out, and an extensive perforation results.

In other instances the sequel is described as a species of *atrophy of the cornea*, which is converted into a whitish, flattened plate (Schmidt-Rimpler).

One or both corneæ may be affected, and the usual cause is exhaustion after acute illness or after prolonged diarrhea or dysentery. A similar softening and sloughing of the cornea may be the result of ophthalmia neonatorum (see page 198), or cataract incisions which have become septic, and xerotic keratitis (see page 262).

13. **Tuberculous Keratitis** (*Tuberculosis and Tuberculous Ulcer of the Cornea*).—Tuberculous lesions of the cornea almost always arise by reason of an extension of this disease from the uveal tract, including the pectinate ligament, and manifest themselves either as tuberculous nodules or as a diffuse parenchymatous keratitis (see also page 266). Primary tuberculous ulceration (Greeff) and tuberculous nodes in the corneal periphery (Bach) have been described which later push their way into the cornea. The lesion not uncommonly has a distinct triangular form (see Fig. 130). A variety of abscess of the cornea, without any healing tendency, which by some authorities has been regarded as a tuberculous process, has been observed in scrofulous children.

Prognosis of Ulcers of the Cornea.—This necessarily depends upon the character and situation of the corneal lesion, but even in the mildest forms some corneal opacity or irregularity of the corneal epithelium will remain (see page 258). If bacteriologic investigation should reveal the presence in the ulcerated area of pneumococci or of a mixed infection, the prognosis is serious, and at once the measures described in paragraph (*b*), page 253, should be instituted in the hope that the spread of infection may be prevented. In severe forms of suppurative keratitis the prognosis is most unfavorable, although active treatment will sometimes be followed by surprisingly good results.

Treatment of Ulcers of the Cornea.—It is not possible to lay down definite rules for the treatment of all forms of corneal ulceration—this must be governed by the exigencies of each case; but certain principles are common to the various types.

Acute Stage: Pain and Photophobia.—These should be relieved by the plans already suggested in the treatment of phlyctenular keratitis. In simple ulcers, atropin, a lotion of boric acid, and dark glasses will usually suffice, and prompt cure often follows an application directly to the ulcer of nitrate of silver (1 per cent.).

Cocain will relieve photophobia temporarily, but its *continuous use in corneal ulceration is positively harmful.* Holocain, on the other hand, is of distinct value, as was pointed out by Hasket Derby, especially if applied directly to the ulcerated surface. If a corneal ulcer is accompanied with much dread of light, the methods described under phlyctenular keratitis may be employed. Dionin (2 to 5 per cent.) is of marked service.

Whenever corneal ulceration is associated with conjunctivitis, the

inner surfaces of the lids may be brushed over with a solution of nitrate of silver, gr. ij–v to ʒj (0.13–0.32 gm. to 30 c.c.), or protargol (5 to 20 per cent.), or argyrol (10 to 25 per cent.) may be freely instilled. The last-named drug is not without danger, as it may cause a permanent brown stain at the seat of the ulcer. The culdesac should be carefully cleansed with a boric acid solution, a collyrium of bichlorid of mercury (1 : 8000), or cyanid of mercury (1 : 2000).

Subacute and Torpid Stage.—After the subsidence of the acute symptoms, or where the ulcer from the beginning is torpid, local stimulation should be secured with an ointment of the yellow oxid of mercury, gr. j to ʒj (0.065–3.885 gm.). Finely powdered calomel dusted into the eye is also of excellent repute. In like manner iodoform or aristol, in salve or powder, may be tried. Eserin has been recommended instead of atropin in small sluggish ulcers.

Deep and Sloughing Ulcers.—It was a universal and is still a common practice to instil a solution of *atropin*, because of its anodyne effect and because it lessens the liability to iritis. The solution should be sterile, as otherwise a simple ulcer may be infected and pass into a sloughing condition.

In some cases *eserin* is employed, because it stops the migration of white blood-corpuscles, or promotes absorption through dilatation of the ciliary vessels, or limits the sloughing process. Furthermore, abnormal intra-ocular tension is lowered by the action of the drug. The strength of the solution may be from ¼ to 1 grain (0.0162–0.065 gm.) to the ounce (30 c.c.), the latter being unnecessarily active in most cases. One or two drops of the eserin solution should be instilled from three to six times daily; and as, under its influence, congestion of the ciliary body and iris may ensue, as well as brow-pain, these complications should be counteracted by using a few drops of the atropin lotion at night. Deep ulcers near the margin are those most suited for the eserin treatment. The author, after considerable experience, is persuaded that eserin in corneal ulceration has a very limited sphere, and that atropin is almost invariably the better drug. Schiötz and Gunnufsen have noted rise of tension in some cases of serpiginous ulcer of the cornea, and think that if it is present mydriasis should be avoided, lest it increase the danger of glaucoma. Atropin, eserin, or pilocarpin may be used in conjunction with dionin, if this drug is indicated, to produce an analgesic or lymphagogue action.

Pain is relieved and the process of repair encouraged by the frequent application of *hot compresses* (see page 201), and by the use of dionin, which may be employed in solution or as an ointment. Hot water (150° F.) dropped directly upon the ulcer is recommended by Lippincott. The culdesac and lacrimal passages must be irrigated frequently with *antiseptic collyria*—a saturated solution of boric acid, bichlorid of mercury (1 : 10,000), aqua chlorini, or cyanid of mercury (1 : 2000).

1. *Impending Perforation.*—When a perforation of the cornea is liable to occur by extension of the ulcer, a *dry antiseptic compressing*

bandage should be applied, removed when the necessary local applications are made, and again reapplied. Long-continued use of the bandage may be followed by eczema of the lids. This should be treated by dusting the parts with calomel or nosophen. Catarrh of the conjunctiva and dacryocystitis contraindicate the use of the bandage unless the danger of perforation is imminent. If dacrocystitis persists in spite of ordinary treatment, the lacrimal sac should be excised (see page 682).

If bulging forward of the floor of the ulcer indicates that perforation threatens, the intra-ocular tension should be lessened by *paracentesis of the cornea*. This operation is described on page 619. It may be necessary to repeat the operation on several days. Intense pain will often be thus speedily relieved and healing rapidly result.

2. *The Spread of Local Infection.*—If, in spite of such treatment, the corneal ulcer continues to spread, either in the form of a lesion creeping across the face of the cornea or by passing inward through its layers, the process must be stopped by one of several means: (1) Scraping with a curet; (2) the direct application of a suitable chemical which combines the properties of a germicide and a caustic; (3) the actual cautery.

(*a*) The ulcer may be curetted with a sharp spoon (under a boric acid spray—de Wecker), all the sloughed material removed, the edges penciled with a sublimate solution (1 : 2000), iodoform dusted upon its surface, and a dry sterile bandage applied. Mules advised softened iodoform wafers.

(*b*) The chemical substances commonly employed are nitrate of silver, carbolic acid, nitric acid, trichloracetic acid, tincture of iodin, and formaldehyd. The first, in the strength of 10 to 20 grains (0.65–1.3 gm.) to the ounce (30 c.c.), is applied directly to the seat of ulceration (care being taken to avoid the surrounding cornea) by means of a probe on which has been twisted a thin band of absorbent cotton, or the point of a pencil of lunar caustic may be gently pressed against the sloughing tissue. Carbolic acid (liquid) may be employed in the same manner as the silver solution; or tincture of iodin, or a caustic solution of formaldehyd (1 : 50), or trichloracetic or nitric acid. Of these substances, carbolic or trichloracetic acid have given the author the greatest satisfaction. Absolute alcohol applied directly to the ulcerated surface is sometimes of great value. Prior to the application of these caustics the ulcerated area should be stained with a solution of fluorescein (see page 50).

(*c*) The actual cautery may be either a small Paquelin or galvanocautery; when neither of these is at hand, a knitting-needle or platinum probe, heated red hot in the flame of a Bunsen burner, will suffice. The edge and floor of the ulcer should be gently but thoroughly burned. Usually one cauterization is sufficient, but in the event of failure to destroy all the sloughing material, the operation should be repeated on the following day (see also page 620). Cocain or holocain render the operation painless, but there is no objection to general anesthesia in nervous patients.

If the surgeon is careful to touch only those portions involved in the

ulcerated process, the resulting scar will not be greater than would have been the case had the ulcer secured cicatrization without such treatment. Fluorescein will show the extent of the ulcer and mark out the area to be cauterized.

The actual cautery may be used to check the advance of sloughing ulcers, although recent improvements in ocular therapeutics have rendered the use of this agent less frequently necessary than in former times. In rodent ulcer (to which it should be applied early and thoroughly) it is one of the few means that is efficacious, and it is also indicated in cases of fascicular keratitis.

Abscess and Hypopyon.—The pus should be evacuated. If the abscess is unbroken, its anterior wall may be incised with a delicate knife, and the subsequent treatment conducted on the principles laid down for sloughing ulcers. If there is hypopyon, paracentesis of the cornea or Saemisch's section (see page 620) may be practised. Subsequently iodoform may be dusted upon the cornea and a bandage applied, to be renewed at suitable intervals.

The antiseptic and specific treatment of ulcers have to a great degree replaced the operation of Saemisch, and in many instances absorption of the products of a hypopyon-keratitis will follow the non-operative treatment already described.

Perforation.—If perforation of the cornea and prolapse of the iris occur, the vigorous use of atropin or eserin, according as the lesion has a central or peripheral situation, aided by gentle effort at *reposition* with a probe, a compressing bandage, and rest in the recumbent posture represent measures which are sometimes followed by success.

In the event of failure, or in any event, if the prolapse is a large one, the iris may be drawn forward through the aperture and excised close to the cornea. After excision the aperture may be covered with a conjunctival flap taken from the bulbar conjunctiva, twice as large as the original opening, into which it is gently inserted with a probe. A firm compressing bandage, not to be disturbed for three days, is applied. This method, which can be recommended, is said by its author, Gamo Pinto, to secure a flat cicatrix, often without any attachment of the iris, although anterior synechia usually results even from the smallest perforation. Conjunctival flaps, formed according to the technic of Kuhnt, may be useful in these circumstances. If the prolapse has been large, a more or less complete staphyloma is apt to follow in spite of vigorous bandaging and the use of eserin or atropin. An early iridectomy may prevent this catastrophe.

Other Methods of Treating Corneal Ulcers.—(*a*) *Dionin.*—The value of dionin in the treatment of corneal ulcer is unquestioned and has been referred to. It would seem that occasionally, in addition to its lymphagogue and analgesic action, it has a positive effect in stimulating corneal regeneration. Immunity is quickly established, and therefore, as a rule, it should be used for three days and then discontinued for two or three days, or until its application is again followed by the dionin reaction. The strength of the solution may vary from 1 to 10 per cent.,

according to the indications, a good average general strength being 5 per cent. Dionin may be combined with atropin, eserin, holocain, and cocain, according to the indications, but, in the experience of the author, furnishes better results if employed in a separate solution, and the other drugs, also in separate solution, are used either before or after its application. Occasionally its action seems to be enhanced by the addition of adrenalin, although adrenalin itself is not a remedial agent of satisfaction. Powdered dionin or dionin in salve may also be used. With its employment by subconjunctival injection, as recommended by some surgeons, the author has no experience. Its advantageous effects are distinctly enhanced by the use of a 2 per cent. solution of holocain.

(*b*) *Serum Treatment.*—Römer, believing that 95 per cent. of infected so-called serpent ulcers are caused by the Fränkel-Weichselbaum diplococcus, has, with the aid of the chemist Merck, developed a serum (*pneumococcus* or *antipneumococcus serum*) which he advises in the treatment of this form of corneal disease. The serum may be used subcutaneously and also subconjunctivally, and may be instilled into the conjunctival sac. Römer himself doubts the value of the subconjunctival injections. Subcutaneously from 3 to 5 c.c. of the serum may be employed, and the results thus far reported indicate that occasionally it seems to facilitate the cure of beginning ulcers, but in large, well-developed ulcers it is ineffectual (Zur Nedden). Complications have been reported—for example, myocarditis and decided febrile reaction are said to have followed the injections (Zeller). With this method of treating corneal ulcers the author has had no experience. According to Axenfeld, this serum possesses curative properties, but, as prepared at present, its action is not sufficiently certain to allow it to replace other methods. Recently Römer has advocated autoserotherapy in the treatment of hypopyon keratitis. The serum obtained from a blister in the patient's arm is injected beneath the conjunctiva, the dose being 1 c.c.

The antistreptococcus serum or vaccine has also been employed in streptococcal infections, but apparently it, like the antipneumococcus serum, should be regarded as supplementary to other forms of treatment. Of staphylococcus serum-therapy too little is known to determine its influence on the eye (Axenfeld). In place of Römer's serum, antidiphtheritic serum has been employed with success (Darier, Zimmermann, Fromaget) in the treatment of severe corneal ulceration. Good results have also followed the use of Deutschmann's serum (yeast serum in the dose of $\frac{1}{2}$ to $1\frac{1}{2}$ c.c. in children, and 4 to 8 c.c. in adults), according to reports by Deutschmann, Von Hippel, and others. In Axenfeld's clinic its effects were negative. In the author's experience bacterins prepared from the micro-organisms responsible for corneal ulceration have proved to be of surprising value in the treatment of infected ulcer of the cornea. These bacterins (or vaccines) have been prepared for him by Dr. B. A. Thomas from patients in the University Hospital. The dose has varied from 50,000,000 to 300,000,000. Such

bacterins represent a therapeutic measure which should receive careful attention and trial.

Hypopyon keratitis has also been treated by means of applications of pyocyanase, prepared from cultures of the pyocyaneus bacillus.

(*c*) *Subconjunctival Injections.*—Naturally, subconjunctival injections, so satisfactory in certain diseases of the eye, have been tried in corneal ulcers. At one time bichlorid of mercury was chiefly employed, but, largely owing to Mellinger's investigations, this gave place to physiologic salt solution, which seemed to act equally well. There is a certain amount of evidence, however, that in infected corneal ulcers cyanid of mercury is the better agent. Of this drug, 10 to 20 minims (0.6–1.25 c.c.) of a 1 : 4000 solution may be injected beneath the conjunctiva; its use, in these circumstances, is strongly urged by Col. Henry Smith. There is no objection, but, on the whole, rather advantage in adding chlorid of sodium to the solution. Of the bichlorid of mercury solution, Dufour has recently recommended injections of 1 : 2000 if the ulceration is active, and 1 : 3000 to 1 : 10,000 if the infection is not very robust. Acoin added to the solutions employed in subconjunctival injections is said to diminish the pain. It should be used in a 1 per cent. solution, one-third of which is added to two-thirds of the solution employed.

(*d*) *Additional Local Measures.*—Reference to the value of iodoform directly dusted upon corneal ulcers has already been made. In its place xeroform or nosophen may be used. Sometimes these remedies are employed in the form of an ointment, as are also iodid of potassium, iodol, and europhen. Iodoform introduced into the anterior chamber after paracentesis in the form of rods, which is so highly recommended in infected wounds of the cornea by some surgeons, is not a useful procedure in hypopyon keratitis. Absolute alcohol applied directly to an ulcerated surface is occasionally of great value. The anilin dyes, in the form of blue and yellow pyoktanin, at one time regarded with favor, in the opinion of the author are valueless. Iodin-vasogen is recommended in infiltrated and spreading ulcers in 0.6 per cent. solution, and ariol dusted on the surface of an ulcer after the application of the actual cautery is recommended by Fischer. If bacteriologic examination should reveal the presence of the diplobacillus in the corneal ulcer (diplobacillary ulcer), the preparations of zinc should be used (sulphate, chlorid, or salicylate in 1 to 5 per cent. solution) and applied directly to the lesion. Zinc preparations are also useful in pneumococcus ulcer, and Morax has tried rabbit's bile with indifferent success. Recently *ethylhydrocuprein* (optochin) has been strongly advocated in the treatment of pneumococcic ulcers of the cornea. It may be applied directly to the ulcer in a strength of from 1 to 2 per cent. by means of a cotton swab held in place for a minute and repeated as required, or used in a watery solution every hour, or employed in the form of an ointment, combined with atropin. Decided improvement has been noted as early as the third or fourth day, and even earlier. At first its application causes much burning pain, which may be checked with holocain. Some

reports of its value are enthusiastic; others express disappointment. Purulent keratitis has been successfully treated by zinc iontophoresis.[1] Darier recommends *scarlet red* in the treatment of infected ulcers (5 per cent. ointment in an aqueous solution), and Haass urges *pellidol* and *azodolen*, derivatives from scarlet red, in 2 per cent. ointment in the management of corneal lesions with loss of epithelium. The application of radium has been advocated by Lawson and McKenzie. In *Mooren's ulcer* of the cornea the use of the actual cautery has received high commendation. H. Lewis Jones has achieved success by using zinc ions. Covering the ulcerated area with conjunctival flaps has been tried; the author has employed this method once with only temporary success. *Thermotherapy* has been employed by Weekers in ulcers of the cornea by means of a platinum filament at a low degree of incandescence, which is held over the surface of the ulcer for one minute without touching it.

(*e*) *Associated Conditions.*—The treatment of conjunctivitis complicating ulcer of the cornea in nowise differs from that suited to ordinary cases. An ulcer should always be carefully examined for the presence of a *foreign body*, which may be covered by a small slough, while *misplaced cilia* are fruitful sources of corneal irritation and may hinder the prompt healing of ulcers. They should be removed with epilating forceps or destroyed by galvanopuncture.

The *lacrimal passages* should be explored and, if strictured, rendered patent, while irrigation of the lacrimal canal with a 4 per cent. solution of boric acid, or 1 : 8000 solution of bichlorid of mercury, or 10 to 20 per cent. solution of protargol or argyrol, is of material aid in the treatment of infected ulcers, because this passage is commonly the seat of unhealthy secretion. If the tear-sac contains pus it should be excised. At the same time the nasopharynx and the accessory sinuses need exploration and treatment of diseased conditions.

The *teeth* should always be examined, and, if faulty, the patient turned over to a competent dentist. The frequent relation of carious teeth and pyorrhea alveolaris to corneal ulceration is well established, and the irritation of a new dentition in young children has been found to be the cause of abscess or ulcer of the cornea. In brief, the entire *cephalic mucous membrane* (Harrison Allen) should be explored, because, in one or other of its component parts, it may be the seat of disease, which, even if it is not the cause of the coexisting corneal ulceration, is none the less responsible for retardation in the healing process. Some corneal ulcers appear to have been caused by disease of the accessory sinuses, especially the ethmoid sinuses, and by purulent rhinitis.

Constitutional Treatment.—The patient, other things being equal, should not be secluded in a dark room, but, with eyes properly protected with goggles, go out into the fresh air every day. The diet must

[1] For the method of applying this remedial agent, see Traquair's article on "The Treatment of Purulent Keratitis by Zinc Iontophoresis," Ophthalmic Review, vol. xxx, 1911, p. 1.

be nutritious and easily digested; tea, coffee, candies, and pastries should be forbidden.

If scrofulosis is present, cod-liver oil, lactophosphate of lime, and iodid of iron or syrup of hydriodic acid are indicated; anemia is best treated with the tincture of the chlorid of iron or with the carbonate of iron; any suspicion of malaria requires the use of quinin and arsenic. The syphilitic taint, which may be present without being the direct cause of the ulcer, indicates the iodids, and mercury, especially in the form of the bichlorid. If ulcers of the cornea occur in gouty or so-called rheumatic subjects, citrate of lithium, mineral waters, iodids, colchicum, salicylic acid, salol, etc., are indicated. Thyroid extract has been used by the author and Veasey in stubborn non-ulcerative keratitis, and this drug has recently again been recommended by Radcliffe. In tuberculous ulceration of the cornea (see page 251) important results are achieved by the administration of tuberculin (see page 318).

A very strict inquiry into the condition of the alimentary canal should never be forgotten. In children, calomel is a useful laxative; in older patients, the salines and saline waters are often necessary.

The urine should be carefully examined for albumin and sugar, and for the products which indicate imperfect assimilation. The influence of enterogenous auto-intoxication must be eliminated.

A very important element in the successful management of cases of sloughing ulcers, especially in subjects of depressed nutrition, is the maintenance of proper circulation; strychnin and digitalis are often indicated. Severe pain may be alleviated by opium or morphin in suitable cases; the drug also has a favorable influence upon the ulceration. Codein also serves a useful purpose.

Results of Corneal Ulceration.—Opacities more or less permanent follow almost all ulcerations of the cornea. If the opacity is slight, it is spoken of as a *nebula* or *macula;* if dense, as a *leukoma.* An old corneal macula possesses a good reflecting surface, which serves to distinguish it, as Haab points out, from a recent inflammatory infiltration, which has a dull surface.

It is evident that upon the position of the opacity in the cornea depends its influence upon vision. The more central it is or, rather, the more directly it encroaches upon the pupillary region, the greater will be the disturbance of direct vision. Inequalities in the curvature of the cornea distort the retinal images and are fruitful sources of irregular astigmatism.

Where perforation has followed ulceration and the iris has remained entangled in the aperture, the attachment is called an *anterior synechia;* the corneal scar to which the iris is fastened receives the name *adherent leukoma* (Fig. 119). An eye thus afflicted may become quiet and retain, either with or without operative interference, useful vision; but may also be subject to recurring attacks of inflammation, and may originate sympathetic irritation or inflammation in the fellow eye. It may also become the subject of glaucoma.

The distention of a cicatrix, to whose inner surface the iris is attached,

constitutes a *corneal staphyloma,* which is called *total* if the entire cornea is involved, *partial* if only a portion is included, and *racemose* if perforations have occurred at various points.

The mechanism of the development of staphyloma is, briefly, as follows: A perforation takes place, and the iris falls forward and at-

Fig. 119.—Adherent leukoma, the result of perforating corneal ulcers (from a patient in the Philadelphia General Hospital).

taches itself to the opening, or protrudes through it, becoming fixed there by the lymph thrown out in the process of repair. The scar tissue which remains fails to withstand the intra-ocular tension, and that portion of the cornea is pushed forward beyond its normal limits, forming a pouch-like deformity.

Fig. 120.—Beginning staphyloma of cornea following perforation of infected ulcer.

The protrusion may flatten down, and under the influence of fresh inflammation bulge forward again, or may extend between the palpebral fissures and prevent the lids from closing (consult Fig. 121). Staphylomas, the result of ulceration, are more or less opaque, because they

represent the scar tissue which has formed after the rupture of the membrane. Corneal staphylomas, which are not opaque and have not formed under the influence of an inflammation, also occur, and will presently be described. Thick corneal scars and staphylomas may

Fig. 121.—Complete staphyloma of the cornea.

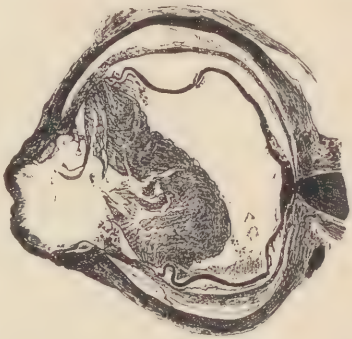


Fig. 122.—Section of an eyeball with complete staphyloma of the cornea.

undergo retrogressive metamorphosis with the deposition of hyaline masses and lime particles in them. Purulent ulcers may develop and perforate the lesion; they may even cause panophthalmitis and subsequent atrophy of the eyeball. Ulcers thus formed were named by Arlt *atheromatous ulcers*. The condition is also called *scar keratitis*, and, according to Fuchs, is due to entrance of bacteria through the diseased and feebly resisting epithelium (see also page 414). Anterior synechiæ and adherent leukoma may also occur, according to Von Hippel, from *internal ulcer*, without suppuration of the anterior corneal layers. Later the eye may become staphylomatous.

Fig. 123.—Staphyloma of cornea, with hypertrophy of the cicatricial tissue.

If after inflammation of the cornea, with loss of its superficial layers, the intra-ocular pressure causes the remaining lamina to bulge forward into an opaque elevation, the condition is called *kerectasia*. This differs from an ordinary partial staphyloma because there has been no perforation, and the iris tissue is not involved in the process.

If all the layers of the cornea down to the posterior elastic lamina are destroyed, and this protrudes through the opening in a small, translucent, hernia-like pouch, surrounded by a rim of opaque cornea, it is known as a *keratocele*.

An orifice remaining after a wound or, more commonly, because of

the failure of an ulcer to heal is designated *fistula of the cornea.* It may last for a long period and stubbornly resist efforts at cure. It has been recommended to touch the mouth of the fistula with a point of lunar caustic, and even to pare the edges and introduce a corneal suture. A cicatrix of horny nature growing from the cornea has been reported by Arnold Lawson, and he thinks the epithelium covering cicatrices may not infrequently become cornified.

Treatment of the Results of Corneal Ulceration.—Satisfactory results follow *massage of the cornea.* The massage movements should be made in a circular and radial manner over the cornea, through the closed lids, after the introduction of a small piece of the yellow oxid of mercury salve into the conjunctival culdesac. Some irritation accompanies the method, but may be allayed by the occasional use of a collyrium of boric acid and cocain. Instead of yellow oxid of mercury salve an ointment of dionin may be used. In place of finger massage, *vibration massage,* introduced by Maklakow, may be employed. An Edison electric pen, the point being armed with a small ivory ball, is employed. The vibration rate varies from 200 to several thousand a minute. Subconjunctival injections of physiologic salt solution may aid in the absorption of corneal opacities following keratitis, and similar injections of magnesium sulphate and sodium sulphate have been recommended. Thiosinamin in ½-grain (0.0324 gm.) doses and gradually increased has been employed in the treatment of opacities of the cornea. The author has tried the remedy without satisfactory results. Other methods of treating corneal scars are as follows: A 5 per cent. solution of a mixture of equal parts of thiosinamin and antipyrin (J. Galezowski); an eye-bath of ammonium chlorid, one to three teaspoonfuls to a cupful of boiled water (Pick); and injections of a 15 per cent. solution of fibrolysin, which is a compound of thiosinamin and salicylate of sodium, the dose being ½ to 2 cm.

Alleman revived the use of *galvanism* for the removal of corneal scars, and reported favorable results. A suitably prepared electrode is connected with a battery, the cathode being applied directly to the anesthetized surface of the cornea, and the anode to the soft tissues of the cheek. Usually a current of from 1 to 1¼ milliamperes gives the best results. The *séance* lasts at the beginning of the treatment for one minute, and is gradually increased to three or four minutes. Great care should be taken not to produce too much reaction. Sulzer recommends electrolysis and phototherapy in the treatment of corneal opacities.

Vision may be improved by an iridectomy for new pupil, and the appearance of the eye by tattooing the cornea with India ink if the corneal leukoma is dense. Attempts have been made at transplantation of rabbit's cornea for the relief of dense central opacities. Some recent efforts at *corneal transplantation* have met with encouraging results, especially if a circle of clear cornea can be removed from a freshly enucleated eye and transferred to the opening made in the leukomatous cornea. Von Hippel's trephine is a useful instrument for this operation.

The treatment of staphyloma in the first place is preventive, and those measures already described in connection with impending perforation of the cornea, and perforation after its establishment, are indicated—namely, a compressing bandage and the use of eserin or, in some circumstances, atropin. If, in spite of this, the bulging continues, paracentesis of the anterior chamber or an iridectomy opposite the clearest part of the cornea may be performed. Indeed, an iridectomy to prevent the formation of staphyloma is often useful while the corneal scar is still flat, especially if the tonometer indicates any rise of tension. If the disease has been so extensive that a complete and unsightly staphyloma has formed, which is the seat of pain and a source of danger to the fellow eye, excision of the globe is indicated, or one of the various substitutes for the operation of enucleation (see page 636).

Keratomalacia (*Xerotic Keratosis*—a name also applied to keratitis e lagophthalmo—*Necrosis Corneæ; Infantile Ulceration of the Cornea, with Xerosis of the Conjunctiva*).—This disease is characterized by dryness of the conjunctiva and destructive ulceration of the cornea, and usually appears in infants during the first year of life. According to Stephenson it is especially liable to arise about the eighth month, but may develop from the third to the twentieth months.

Cause.—It occurs only in anemic, badly nourished individuals. It has been seen accompanying meningitis, measles, and variola, and among children with diarrhea, enteritis, tuberculosis, syphilis, and those who are inmates of homes whose surroundings are unhygienic. Bacilli have been found, but the special microbe, if it exists, has not been certainly isolated. In a few cases the Spirochæta pallida has been found (Stephenson). The disease is not a common one. A somewhat similar condition has been described in the eyes of negro children in the South (Kollock).

Symptoms.—In the beginning there are conjunctival congestion and lacrimation, but the peculiarity of the disorder is the development of the appearances described under Epithelial Xerosis (see page 230), in connection with the corneal lesions. A gray haze, rapidly turning into ulceration, appears in the cornea, followed by inflammation of the iris and the formation of hypopyon. Perforation of the cornea and destruction of the eyeball may result. Both eyes, as a rule, are affected, one earlier than the other.

The *prognosis* is very unfavorable; the patients usually die (according to Stephenson the disease is fatal in 50 per cent. of the cases) of the wasting disease which has occasioned the trouble, or of an intercurrent pneumonia. In some cases streptococci have been found in the local lesions, and foci of these micrococci scattered throughout the body.

Treatment.—This resolves itself, besides the ordinary treatment of severe corneal ulceration, into the administration of the internal remedies which are indicated by the general state of the patient.

Neuroparalytic keratitis is the name applied to an ulceration of the cornea which arises when this structure becomes anesthetic, because it is severed from the influence of the trigeminus.

Causes.—Disease of the Gasserian ganglion, or its removal for trifacial neuralgia, disease of the nuclei of the fifth pair, periostitis of the orbit, syphilitic deposits, and fracture of the skull may cut off the trigeminal influence and cause the affection.

The corneal lesion has been ascribed to a trophic change; to the lessened power of resistance which the cornea in its insensitive condition presents to external injuries; to the irritation of the fifth nerve by the lesion; to micro-organisms; and to increased evaporation from the surface of the cornea.

Wilbrand and Saenger dismiss the traumatic theory and believe that some trigeminal fibers from the first branch remain and carry the irritation, which is finally concerned with the development of the disease. Such irritations, according to Verhoeff, might readily cause an acid reaction and, if sufficiently long continued, originate the lesions. Hence, in the language of Parsons, the disease is probably due to irritative changes in and about the degenerating nerve.

Symptoms.—The keratitis begins in the true corneal tissue, and spreads peripherally until the central necrosis or slough separates, and perforation of the cornea with prolapse of the iris occurs. The anterior chamber may contain pus or pus mixed with blood. Beyond and around the central abscess the corneal tissue is comparatively clear, especially in a margin of 2 to 3 mm. in width, but in the periphery there may be secondary foci of infiltration, closely connected with inflammation of the neighboring conjunctiva. The surface of the cornea and conjunctiva is anesthetic. The intra-ocular tension is diminished. There may be considerable pain and irritation or these symptoms may be absent.

The *prognosis* is unfavorable, and in spite of treatment destructive inflammation often results, although occasionally the keratitis subsides without the formation of purulent material. The center of the cornea, however, is flattened and presents a dense scar at the termination of the disease.

Treatment.—The usual treatment of corneal ulcers is necessary, and the affected eye should be excluded from the influence of external irritants, either by a carefully applied antiseptic bandage or by a Buller's shield, or, better, by stitching together the lids. The same operation is indicated as a prophylactic measure preceding removal of the Gasserian ganglion. At least, the eye should be covered with a protecting shield, such as has been devised by W. W. Keen. Experimental evidence indicates the propriety of preventing evaporation by keeping the eye in a moist atmosphere.

Keratitis e Lagophthalmo (*Keratitis of Desiccation; Keratitis Xerotica*—Feuer).—This affection arises because the cornea is exposed, owing to defective closure of the lids (see also pages 180 and 584). As the result of this exposure there is desiccation of the corneal epithelium, which becomes fissured and in places exfoliates. Thus, a pathway for microbic invasion is opened, and ulceration and suppuration occur. The usual *causes* of this affection are: proptosis of the eyeball, as in ex-

ophthalmic goiter (see page 583) and exophthalmos (see page 592); paralysis of the orbicularis, as in facial palsy (see page 180); and long-continued illness associated with defective closure of the lids.

The **treatment** consists in protecting the cornea by a suitable bandage or shield, or in some cases by stitching the lids together (see page 604).

Herpetic Keratitis (*Herpes of the Cornea*).[1]—The corneal lesions associated with herpes zoster ophthalmicus have been described on page 163. The present disease consists of a vesicular eruption upon the cornea, which breaks down and forms an ulcer, characterized by a denudation of epithelium not unlike that produced by injury.

Causes.—Horner described herpes of the cornea with whooping-cough, intermittent and typhoid fever, and, in general terms, with those affections in which herpes of the lips and nose is found. It is seen in acute and subacute disease of the posterior nares and pharynx, and also in affections of the respiratory apparatus generally (pneumonia, bronchitis), and may follow or be associated with influenza.

Symptoms.—The disease begins with the symptoms of catarrhal conjunctivitis followed by a series of transparent vesicles upon the cornea, which have been compared to a string of small beads. The vesicles are placed in a circle, or run in a diagonal or irregular line

Fig. 124.—Showing various shapes and positions of herpes ulcers (Haab).

across the cornea. They speedily rupture and leave an open patch, deprived of epithelium, which is anesthetic and has irregularly serrated margins, upon which the remains of vesicles may be seen. The lesions are easily shown by fluorescein, which may also produce a deep or superficial coloration of those portions of the cornea apparently unaffected. The sensation of the cornea is diminished.

The progress of repair is slow, and is often interrupted by the reappearance of fresh vesicles. The disease may be complicated with pus in the anterior chamber and iritis. Pain in the eye and brow, photophobia, lacrimation, and a gritty sensation are the subjective symptoms.

Treatment.—This consists in relieving the general condition; usually quinin in full doses is indicated, and salicylate of sodium is a most valuable remedy. During the stage of irritation, atropin, holocain, warm compresses, and dark glasses are needed. Dionin is of signal service. After the formation of the ulcer the treatment is conducted on general principles. A pressure bandage is of advantage, and in many cases an application of tincture of iodin is promptly successful. It may be repeated as often as required. Occasionally the application

[1] This term, as Horner observed, is often incorrectly used as synonymous with phlyctenular keratitis.

of carbolic or trichloracetic acid or the actual cautery may be needed to subdue stubborn ulcers of this character.

Rosacea Keratitis.—Usually in women at or about the fortieth year of life, but sometimes at an earlier period, in association with acne rosacea, there may develop a form of keratitis. The manifestations vary. There may be lacrimation, blepharospasm, blepharitis, bulbar injection, and the development of a grayish-white, vascularized corneal infiltration with small circular ulcers, as in Holloway's patient; or small marginal ulcers and infiltrations may arise (Erdmann; see also page 189). The treatment of the coexisting acne rosacea is important. The eye lesions are favorably influenced by the usual lotions, by scopolamin-mydriasis, and especially by holocain.

Keratitis bullosa in many instances is a symptom and not a separate disease, inasmuch as it consists of the formation of one or more small blebs of short duration (*keratitis vesiculosa*), or of larger blebs of more enduring existence (*keratitis bullosa*), upon the cornea of an eye the subject of iridocyclitis, interstitial keratitis, or glaucoma.

Cause.—This affection formerly was attributed to a mechanical effect due to increased intra-ocular tension. Probably under the pathologic conditions existing an interepithelial edema takes place, the fluid penetrating from the anterior chamber through the changed and unresisting endothelial cells, or coming from the capillary network of the corneal limbus. This edema causes the epithelial cells to degenerate and loosen their hold on Bowman's membrane, and they are raised in the form of bullæ. Sometimes, in addition to epithelium, the walls of the bullæ are composed of a homogeneous membrane. Occasionally moderately large vesicles form upon a cornea otherwise normal, and in one reported case malaria was believed to be the chief factor in their causation.

Symptoms.—In addition to the formation of the blebs, there are burning pain, photophobia, injection of the bulbar conjunctiva, and rupture of the vesicles, leaving an abrasion which may go on to ulceration, and its infection may produce sloughing of the cornea, and even panophthalmitis. There is a strong tendency to recurrence, and with each new formation of vesicles the violent inflammatiory symptoms are repeated.

Treatment.—This consists in puncture of the blebs and suitable local measures, according to the causative disease. Holocain, dionin, and sometimes pilocarpin are useful. In severe cases iridectomy and even enucleation may be needed. The recurrent character and the remissions which have been described have suggested the use of antiperiodic doses of quinin; and these have been given with good results.

The second group of corneal inflammations, a description of which follows, is the *non-ulcerative*, and includes a variety of affections free from ulceration.

Vascular keratitis is a superficial vascularity and opacity of the cornea, and is seen in pannus caused by granular lids (see page 221) and in phlyctenular pannus the result of many relapses of phlyctenular keratitis (see page 242).

Another form of vascular keratitis is characterized by the formation of two opposite vascular areas at the upper and lower margins of the cornea, which approach each other until the vascularization is complete. The disease is met with in young adults and in unhealthy, scrofulous, and underfed children. The second eye usually is attacked, and, as was pointed out by Carter, the character of the disorder indicates a perverted action of the nerves which govern the areas affected, and places it in analogy with herpes.

Symptoms.—These begin insidiously with slight intolerance of light, preceding the appearance at the upper margin of the cornea of a crescent of closely arranged blood-vessels, which, as they advance, push before them a border of corneal opacity. Simultaneously the same appearances become manifest at the lower margin. Between the vascular areas the cornea is grayish white in color, or it may assume an appearance which suggests a purulent infiltration of the layers. Clearing begins at the borders, and the whitish opacity which remains leaves the center last of all. All cases must be regarded with anxiety, as the lesions do not always entirely disappear (compare page 270).

Treatment.—Local irritants are contraindicated. Atropin, dionin, and warm fomentations in the early stages, and later a salve of the yellow oxid of mercury or calomel are useful. The best internal treatment is a prolonged course of iron and bichlorid of mercury. Iridectomy for new pupil may be necessary, and the convex side of the vascular crescent may be touched with the galvanocautery, or a free *peritomy* may be performed.

Parenchymatous Keratitis (*Interstitial, Syphilitic, Inherited, Specific, and Diffuse Interstitial Keratitis; Anterior Uveitis*).—This is a diffuse keratitis in which a chronic inflammation of the whole thickness of the cornea takes place, until, usually without ulceration, but always with superficial or deep vascularization, the cornea in severe cases passes into a condition of universal thick haziness.

Causes.—The majority of cases of interstitial keratitis are due to *inherited syphilis,* the evidence of which is present in 60 per cent. of them. In a comparatively small percentage of cases (2 to 10 per cent., according to Stephenson's investigations) *acquired syphilis* is the etiologic factor. Next to syphilis, tuberculosis furnishes the largest contingent of cases of interstitial keratitis (about 10 per cent.). This disease may also be caused by rachitis, scrofula, malaria, myxedema, trypanosomiasis, leprosy, the climacteric, and depressed nutrition. It is occasionally seen in animals and may be the result of trauma. Its causation by syphilis, tuberculosis, leprosy, and trypanosomiasis has been proved not only in clinical, but in histologic, bacteriologic, and experimental investigations.

It is most frequently observed between the ages of five and fifteen years (most frequent between six and twenty—Hoor), occasionally as early as the third year, but rarely after the thirtieth year. A few cases are on record as late as the sixtieth year of life. The disease is

more frequent in females than in males, occurring in the former especially at the periods of second dentition and of puberty.

Interstitial keratitis appears to have been aggravated by the development of menstruation, and also to have undergone improvement by establishment of the menstrual molimen. It is probable that the affection occasionally arises *in utero,* and a *congenital form* of interstitial keratitis, not differing in appearance from the ordinary or postnatal form of the disease, has been described (Randolph). It has been produced in animals by inoculation of the eye with syphilitic material, and in human beings it has followed a chancre on the lid or conjunctiva (10 cases, according to J. T. Carpenter). Fuchs has observed in some cases of interstitial keratitis that in shape the cornea is a vertical ellipse. In general terms he believes that an *oval cornea* is more frequent in persons with inherited syphilis and in those who are likely to acquire interstitial keratitis than it is in other patients. It does not follow, as he points out, that because the cornea is oval the patient has inherited syphilis.

Fig. 125.—Vessel formation in the cornea after interstitial keratitis (Hirschberg).

Symptoms.—The lesions begin either in the center or at the margin of the cornea. In the first instance, after a few days of slight ciliary congestion and watering, a faint cloudiness appears. The spots of haze, if carefully examined, will be found to be interstitial opacities, composed of round cells—that is, within the structure of the cornea itself and not on either surface.

In two or three weeks they spread until the whole cornea is invested with a diffuse haziness, veiling or completely hiding the iris, except, perhaps, through a narrow rim at the margin of the cornea. The steamy surface has often been compared to ground glass. Careful inspection will reveal that the opacity is not uniform, but contains saturated whiter spots scattered through it, which have been described as "centers of the disease." There are always at this stage ciliary congestion and some pain and dread of light. Blood-vessels derived from the ciliary vessels are thickly set in the layers of the cornea and produce a dull red color—"the salmon patch of Hutchinson." These patches may be small and crescent shaped, or large and sector-like. In one type (referred to on page 266) the vascularity creeps from above and below until the entire cornea is cherry red. If the disease begins at the *margin* of the cornea, areas of cloudiness appear at different portions of it, and gradually from all sides approach the center until the general haziness is complete. Owing to the formation of vessels, the limbus becomes red and swollen at those portions which correspond to the marginal opacities, giving rise to an appearance which has received the

name "epaulet-like swelling." It is most often seen in the upper corneal margin.

The subjective symptoms of irritability and photophobia are more pronounced in strumous children who are at the same time syphilitic. Ulceration rarely occurs, but none the less ulcers of discoverable size are sometimes present, and hypopyon and an appearance resembling an

Fig. 126.—From a photograph of a patient in the Children's Hospital, the subject of inherited syphilis and interstitial keratitis.

accumulation of pus in the layers of the cornea have been reported. Iritis and the formation of posterior synechiæ are not uncommon, in one form the iritis being associated with deposits on the posterior layer of the cornea and the formation of anterior synechiæ. Inflammation of the ciliary region is occasionally encountered; secondary glaucoma and shrinking of the eyeball may follow.

In the course of time, varying in accordance with the treatment,

the eye begins to clear, usually from the periphery. Perfect recovery of the transparency must be rare, although the remaining haze may be slight. Years after an attack of interstitial keratitis minute vessels, nearly straight, branching at acute angles and short bends, may be detected in the cornea. These appearances have been especially described by Nettleship and Hirschberg, the latter observer stating that the vessel formation never subsides entirely, and he has seen this condition, with the aid of a corneal loup, thirteen years after an attack.

In addition to the complication of iritis and inflammation of the ciliary body, more or less retinitis is very apt to be present. Disseminated choroiditis, and even optic neuritis and retinal hemorrhage, have also been observed; indeed, it is not uncommon to find, far forward in the eye-ground, areas of choroiditis (anterior choroiditis) not only in the diseased, but also in the unaffected eye (see also page 351). The presence of the vessels and the deposits in the retina and choroid after the disease has subsided may be utilized for the diagnosis of inherited syphilis. Secondary glaucoma may develop, with deep cupping of the disk. Periods of rise of tension during the course of interstitial keratitis are not uncommon, and must be carefully guarded against, using eserin or by paracentesis of the cornea.

The subjects of typical forms of this disease often present a remarkable combination of physical defects. The dwarfed stature, the coarse, flabby skin, the sunken nasal bridge, the scars at the angle of the mouth and also of the nose, the malformed permanent teeth, in which the central incisors have vertically notched edges (Hutchinson's teeth), indelibly stamp the inheritance of the patient. This character of teeth is present in between 20 and 30 per cent. of the cases. Indeed, it has been seen as frequently as 31 times in 48 cases. The presence of deafness, cicatrices in the pharynx, chronic periostitis of the tibia, synovitis of the knee-joint (symmetric or unilateral), and indurated lymphatic glands further emphasize the syphilitic taint. Not only are the different forms of Hutchinson's teeth frequently evident, including the peg-shaped milk canine, but also the defective first permanent molars described by Fournier and Darier, and the "sloped molar" of Gifford.

The interstitial keratitis of *acquired syphilis* is usually a late secondary or a tertiary event. It may be circumscribed or diffuse, and is more apt to be unilateral than the variety due to inherited syphilis. Its evolution is relatively more rapid, and it is more promptly amenable to treatment. Usually it appears in adults between the twentieth and fiftieth year of life; exceptionally it has been seen in children (see also page 267).

Diagnosis.—The course of the disease is usually quite typical, and the associated symptoms characteristic. The tension of the eyeball and the age of the patient in most instances help to exclude primary glaucoma, while the history and character of the inflammation differentiate it from old corneal maculæ and from the diffuse infiltration of the cornea which is sometimes seen as the result of injury. Wassermann's test should always be made, and, to distinguish between cases

due to syphilis and tuberculosis, the reaction of the patient to tuberculin or to Von Pirquet's test should be tried.

The presence of the minute straight vessels is good evidence of former interstitial keratitis. These vessels must be distinguished from those which remain after pannus from granular lids. According to Hirschberg, in the latter condition they are more superficial and pass into anterior conjunctival vessels. There are well-formed anastomoses, the broader veins are accompanied by finer arteries, and there are peculiar ramifications of the small deep vessels. The vessels seen in corneal scars after ulceration are confined to these cicatrices. The rest of the cornea is free.

Certain atypical cases of interstitial keratitis have been described, namely, forms in which the opacities are *stripe-like;* others in which they are *ring-like;* others presenting the appearance of pus in the layers of the cornea, the so-called *abscess forms;* others in which there is a combination of *interstitial keratitis* and *keratitis punctata,* and that form which is spoken of as *central annular interstitial keratitis,* especially described by Vossius, and usually seen in individuals under the age of twenty, and for which a definite cause has not been found. The variety which begins as a *marginal vascular keratitis* has been described. Corneal ulceration is uncommon, but is occasionally encountered.

Prognosis.—From six to eighteen months are usually consumed in the development of the various stages of the disease. The second eye is almost certain to be attacked in from a few weeks to two months; even active medication may not prevent the involvement of the second eye. In rare instances the interval is many months, even a year; it may be delayed from five to six years. The patient or his friends must be warned of this fact.

A return to perfect transparency is unusual. The vessel formation in the cornea probably never subsides entirely, but even long-continued opacity in the course of time may markedly lessen, and reasonable vision be restored. The occasional onset of deep-seated inflammation of the ciliary region, and the fact that after the cornea has cleared evidences of former choroiditis, retinitis, or disease of the optic disk, with glaucomatous cupping, may be discovered, must not be forgotten in rendering a prognosis.

Relapses are frequent (18 to 22 per cent. of the cases, according to Hoor), not only of the corneal disease, but of the complications found in the iris and retina. Von Szili suggests that the causation of relapses may be due to *anaphylaxis,* to which he also, in part, attributes the development of the disease after traumatism. It has been taught by some observers that the disorder is more severe now than in former times.

Pathology.—The principal changes occur in the deeper layers of the substantia propria of the cornea, and consist essentially of dense infiltrations of these areas. Newly formed blood-vessels are seen in the posterior and middle layers, and there may be nodular collections of lymphocytes (Fuchs). Some authorities distinguish between *primary* and *secondary* interstitial keratitis, the latter being associated with

inflammation of the uveal tract. Leber regards the disease as always secondary to a uveitis, and this view is strengthened, according to Parsons, by reason of the frequency with which anterior choroiditis can be found ophthalmoscopically in the less affected eye, and by such microscopic examinations which have been made. Some authors (Von Hippel, Elschnig, Stock) believe that this parenchymatous keratitis is primary—*i. e.*, that the corneal disease is the direct result of the general infection. Other observers (Stephenson) ascribe the corneal disease to an extension of the process from the uveal tract; that is, in syphilitic cases, the spirochetes, arriving from this area, proliferate in the corneal tissue. Referring especially to syphilitic interstitial keratitis, two views in regard to the nature are maintained, either that it is due to the direct action of the spirochetes which have been found in the cornea (Von Hippel, Clausen, Igersheimer), or that it is an indirect manifestation of syphilis, *i. e.*, a para- or meta-syphilitic affection. Von Hippel, in some histologic investigations, found nodules in the cornea containing epithelioid and giant cells. Injections of *tuberculin T.* cause a local reaction in some of the cases, which suggests a tuberculous nature of the process.

Anaphylactic keratitis has been produced by Wessely, Von Szili, and Arisawa which closely resembles human interstitial keratitis. Thus, Wessely obtained these results by injecting 2 drops of sterile horse serum between the lamellæ of one cornea, followed in fourteen days by a similar injection between the lamellæ of the other cornea. Von Szili and Arisawa produced opacity and vascularization in a cornea sensitized fourteen days previously by means of an injection into the auricular vein. It is not impossible that interstitial keratitis should be regarded as an anaphylactic phenomenon. Thus, Derby, Walker, Igersheimer, and Schoenberg reason that the spirochetes or their toxins may sensitize the cornea during extra- or intra-uterine life. If later, during childhood, a new amount of latent syphilitic virus enters the previously sensitized cornea, keratitis results.

Treatment.—All irritating applications are harmful. Atropin, to maintain mydriasis, prevent iritis, and allay inflammation, should be systematically employed, unless rise of tension appears, when it must be discontinued. Dionin is of distinct service. The frequent use of hot fomentations is useful, and tenderness in the ciliary region will be relieved by a leech applied to the temple. The eyes may be protected from dust and light by goggles or a dark shade.

A long-continued course of mercury is indicated. The most satisfactory method of administration in the earlier stages is by inunctions, 1 dram (3.885 gm.) of the ointment rubbed into the skin once or twice a day, according to circumstances. Mercury with chalk, 1 grain (0.065 gm.) three times a day, is highly recommended. Subconjunctival injections of bichlorid of mercury have been advocated, but in the author's experience have proved an unsatisfactory method of administering mercury in this disease. Similar injections of cyanid of mercury have been employed. Injections of saline solutions are

often of decided advantage. L. Webster Fox advises subconjunctival injections of sodium saccharinate (1 to 3 per cent.). Some surgeons recommend that mercury be given in the form of hypodermic injections. An experience with this plan of treatment has not caused the author to abandon the usual methods of administration.

During the time the inunctions are being employed, cod-liver oil may be exhibited; later, bichlorid of mercury is a valuable remedy, and, as many of the patients are anemic, this is advantageously combined with the tincture of the chlorid of iron. Arsenic is useful, and atoxyl is highly recommended by Stephenson in doses of 0.25 to 0.50 gram, injected beneath the muscles of the back once or twice a week. A course of tonic treatment, nourishing diet, exercise, and healthful surroundings are necessary; in short, all measures are indicated which elevate the standard of the patient's general health. Indeed, it is most important to treat the subjects of this disease most carefully from the dietetic standpoint. Injections of *tuberculin T.* in those cases depending upon tuberculosis have proved to be efficient. They should be administered according to the methods described on page 318.

Much difference of opinion exists as to the value of *salvarsan* and *neosalvarsan* in the treatment of interstitial keratitis of luetic origin. In the author's experience this form of medication is most valuable, and in syphilitic patients it quickly causes a subsidence of the irritative phenomena. The injections of neosalvarsan are given at intervals of one to two weeks; usually small doses are employed. During the intervals the medication should consist in mercurial inunctions, iodid of potassium, or mixed treatment. The dose of salvarsan must be regulated according to conditions and symptoms; usually each dose should be 0.4 gram. Sometimes 0.2 gram will suffice.

When all irritation has subsided, clearing of the remaining opacity is facilitated by the use of a salve of the yellow oxid of mercury, together with massage of the cornea, or by the local use of a solution of iodid of potassium. Subconjunctival saline injections may facilitate the absorption of the corneal opacities. Iridectomy, if the tension rises and glaucoma threatens, may be necessary; it is evident that it should be employed for new pupil if a stubborn central opacity remains.

Keratitis Punctata Syphilitica (*Keratitis Punctata Vera* [Mauthner]; *Keratitis Interstitialis Punctiformis Specifica* [Hock]; *Keratitis Punctata Profunda* [Fuchs]).—This form of keratitis was originally described by Mauthner, and is characterized by the appearance of circumscribed, pin-head sized grayish spots in the parenchyma of the cornea; episcleral injection is usually wanting. The iris is not involved, the overlying cornea appears transparent, and the dots may arise quickly and disappear rapidly without leaving a trace. They probably indicate a gummatous infiltration of the cornea. This disease is a rare manifestation of syphilis in its later stages, and should be treated with the usual antisyphilitic remedies.

Keratitis punctata is characterized by a precipitate of opaque dots, generally arranged in a triangular manner, upon the posterior

elastic lamina of the cornea (Descemet's membrane—hence also called *descemetitis*). The overlying cornea is hazy, its surface at times slightly uneven. This affection is always secondary to disease of the iris, ciliary body, choroid, or vitreous, and hence is a symptom and not a specific disease. It will be fully considered elsewhere (see page 323).

Keratitis Profunda (*Central Parenchymatous Infiltration; Circumscribed Parenchymatous Keratitis*).—This form of keratitis is characterized by the formation of a grayish opacity in the deeper layers of the cornea, sometimes without severe irritative symptoms and unassociated with ulceration.

The *cause* is not always discoverable; sometimes alcoholic excess, cold, rheumatism, and malaria may originate the disorder; it undoubtedly may develop from an injury. W. T. Holmes Spicer believes that overeating and drinking, and their results in the individuals or in their descendants, in the form of gout and rheumatism, with defective intestinal functions, are responsible for the majority of the cases. It is probably an expression of gastro-intestinal auto-intoxication in some cases. An *acute interstitial keratitis* in association with mumps has been described.

The following is Fuchs' description of this disease: The gray opacity, usually in the center, is covered by the superficial corneal layers, which are hazy and stippled, but not absorbed. Close examination (with a loupe) of the corneal opacity resolves this into individual points, spots, or gray interlacing stripes. The deposit slowly absorbs without ulceration, and commonly with only slight vesicle formation, and leaves the cornea clear, or permanent opacity may remain. Symptoms of inflammation may or may not be present; there is hyperemia of the iris. The duration of the disease is from one to twelve months, the average duration being about three months. Spicer thus summarizes the symptoms of *deep keratitis:* Moderate ciliary congestion, moderate vascularization, but no salmon patch, opacification of the cornea, either as a central disk or a peripheral cone, an appearance under loupe examination of fine striated lines, and edema of the cornea. Fluorescein causes the deepest parts of the cornea to take on a stain. He believes that the true seat of the disease is in the nutrient blood-vessels.

The *treatment* requires atropin, dark glasses, and, later, yellow oxid or similar salve to aid resolution. Dionin and subconjunctival injections of salt or cyanid of mercury may be tried. The constitutional treatment is most important, and is governed by the probable cause.

Among the more uncommon forms of corneal inflammation the following may be mentioned:

Keratitis Superficialis Punctata (*Keratitis Subepithelialis Centralis; Keratitis Maculosa; Noduli Corneæ; Relapsing Herpes Corneæ*).—This disease appears in several forms, just as it has been described under several names, either different types of the same disorder or closely analogous manifestations.

Generally it begins with the symptoms of a sharp conjunctivitis in

which the secretion is watery, while at the same time there is catarrhal disease of the respiratory tract. In two or three days numerous small punctiform or linear spots appear, not immediately beneath the epithelium, but below Bowman's membrane. The overlying cornea is slightly hazy, and the epithelium above the spots a little elevated, the foci being more numerous near the center of the cornea than at the periphery. The cornea intervening between the spots is somewhat hazy, and contains small points and gray lines radiating hither and thither, comparable to the fine fissures in ice. The disease is tedious and may last for months. It occurs in young individuals, usually is bilateral, and is unaccompanied by loss of epithelium, ulcers, iritis, or hypopyon.

Stellwag described foci of large size, most commonly in the periphery. This type of the disease begins with pain in the brow, and the iris may be involved (*nummular keratitis*). It is analogous to interstitial forms of keratitis. In his cases the duration was much shorter, cure having been effected in two weeks.

The anatomic nature of the spots is uncertain; by some observers they have been believed to be enlarged and opaque corneal corpuscles, or lymph-spaces filled with opaque matter. The disease may be associated with catarrhal affections of the upper air-passages. It has been observed in association with menstrual disorders (Bosser), but its exact nature is unknown. Verhoeff's investigations lead him to regard the affection as a neuroparalytic keratitis, with the causal lesion in the ciliary ganglion. The infiltrations beneath Bowman's membrane he attributes to the action of pyogenic diffusible toxic substances arriving at nerve terminals. The disease differs from herpes in the absence of vesicle formation and herpes of the face, in its bilateral character, and in the great number of corneal spots or foci.

The *treatment* should be directed to the mucous membrane of the nasopharynx as well as to the eye. Locally, during the state of irritation, atropin is indicated, and later yellow oxid salve. Holocain and dionin are of service. Full doses of quinin would seem to be called for, and it has been recommended to use the constant current along the region of the distribution of the supra-orbital nerve.

Keratitis Marginalis Profunda.—Under this name, which is here used in a sense quite different from that employed on page 242, Fuchs has described a rare form of keratitis in which a yellowish-gray zone of opacity, immediately joining the sclera, pushes into the clear cornea, accompanied by severe inflammatory symptoms, and occupies about one-half of the corneal circumference. The vessels of the limbus cover the opacity; in several weeks these and the inflammatory symptoms subside, leaving a rim of infiltration somewhat like an *arcus senilis*, save only that it joins the sclera directly and is not separated from it by a stripe of clear cornea. The disorder is unaccompanied by ulceration.

It should not be mistaken for the angular corneal opacity, which appears in connection with scleritis, and which is known as *sclerotizing*

keratitis (see page 290); it differs from it in the absence of any preceding scleritis. The disorder occurs in elderly subjects.

Keratitis Disciformis (*Keratitis Annularis et Disciformis*).—According to Fuchs, this is an individual type of ring-like or disk-like keratitis. It has also been described by Grunert. It should be distinguished from the annular keratitis of Vossius (see page 270). The disease is found in persons in middle life, and appears frequently after slight epithelial defects, whether these are caused by injury or by herpes of the cornea. It is characterized by a delicate gray disk which occupies nearly the middle of the cornea, and which is separated from its transparent margin by an intensely gray, sharply marked border. The superficial layers of the cornea are smooth and unirritated. In the course of the disease, which lasts usually for several months, small ulcers may appear, and under most circumstances there is a decided opacity after the subsidence of the disease. Fuchs thinks that this disease has a position between serpiginous ulceration and the flat, disk-shaped ulceration after herpes of the cornea. All three depend upon an infection of the cornea which gains entrance through a breach in the epithelium. The difference depends upon whether there is a deep or a superficial involvement of the tissue, which, in its turn, depends probably upon the character of the bacteria. Peters emphasizes the connection between this form of keratitis, corneal erosions, and serpent ulcer, and believes that all three depend upon a nervous lesion affecting the corneal epithelium, followed by edema of the tissue. Verhoeff contends that the disease is neuropathic in origin. Schirmer has described circumscribed parenchymatous keratitis, exactly resembling keratitis disciformis, due to infection with vaccine virus. The *treatment* may include atropin, hot compresses, and the local application of absolute alcohol, but it has not been followed by encouraging success. Dionin may be tried.

Grill-like Keratitis or Corneal Opacity (*Gittrige Keratitis*, Biber, Haab); **Nodular or Guttate Opacities of the Cornea** (Groenouw, Fuchs); **Family Punctate Degeneration of the Cornea** (Fehr).—Grill-like corneal opacity, known also under the name of *trellised and lattice-form opacity of the cornea*, was first described by Biber and Haab, and has been well investigated by Freund. The last-named author gives to these opacities the following characteristics: They constitute a hereditary disease which appears first after the age of puberty, in the form of gray, superficially placed spots in and around the center of the cornea, which lie beneath the epithelieum and lift it into a position of distinct unevenness on the superficial layers of the cornea, and by diffuse corneal opacity which, examined with a loupe, is seen to be composed of a grill-like network with radial opacities. The peripheral borders of the cornea are free from disease.

Nodular or guttate opacities of the cornea were first described by Groenouw and later investigated by Fuchs. According to these authors, the disease consists in the development of numerous small, rounded, or irregular gray opacities in the cornea, especially within the pupillary

area. Between the larger opacities lie much smaller, dust-like gray points. The epithelium is slightly raised by the larger nodes, and, therefore, there is a certain slight irregularity of the corneal surface. Almost all of the cases have occurred in men, and they were not found to be associated with any constitutional disease. In some of the patients a history of previous corneal inflammation was obtained. *Reticular opacities* and *interstitial punctate opacities* are names also suggested for this disease.

With the name "family punctate degeneration of the cornea," Fehr has described a punctate opacity of the cornea which may affect several members of one family, and which begins about the tenth or twelfth year of life, progressing steadily until toward middle life. The cornea presents a diffuse gray appearance, and is strewn with white spots and dots of various shapes in the center, while the periphery remains comparatively clear. With a strong lens the diffuse opacity is seen to be composed of minute points, by the condensation of which the larger opacities are formed. The corneal surface is smooth, reflects evenly, and has normal sensibility. Although this corneal condition differs somewhat from the two previous ones just described, it evidently is analogous, as Fehr points out, to them, and he suggests that these lesions probably represent three different types of the same affection, for which he proposes the name "family punctate or spotted degeneration of the cornea."

These various processes represent a degeneration rather than an inflammation, and the opacities are probably due to deposits of hyalin material in the deeper layers of the corneal epithelium and in Bowman's membrane. There may also be a mucoid substance produced by degeneration of the corneal lamellæ. Nodular opacities of the cornea (Groenouw) are regarded by Wehrli as a form of chronic tuberculous disease of the anterior layers of the cornea (*lupus of the cornea*). Treatment is absolutely unavailing.

Marginal Degeneration of the Cornea.—This is probably similar to, if not identical with, the *furrow-keratitis* of Schmidt-Rimpler, and has been well studied by Fuchs, George Coats, and a number of observers. It occurs in middle-aged or elderly persons in whom usually an arcus senilis becomes wider and the cornea in the area of the arcus grows thin. A groove or gutter forms; this gives way before the intraocular tension, and a *marginal ectasia* is developed. The symptoms of irritation are mild; sometimes the lesions are unilateral, sometimes bilateral. In Zentmayer's patient the grooves encircled the margin of the cornea of each eye except for an arc about 15 degrees down and in. Whether the process is degenerative or inflammatory has not been decided. Terrien has tried the application of the actual cautery for its relief.

Epithelial Dystrophy of the Cornea.—According to Fuchs, this degenerative disease of the cornea affects only elderly persons, being more common in women than in men; it has also been noted in association with tabes dorsalis by Fuchs. The corneal sensibility diminishes,

a diffuse opacity of the cornea develops in the pupillary area, associated with marked alterations in the epithelium, the surface of which is uneven and shows blebs or small dark spots. While the epithelium is the chief site of the lesion, there is also stippling in the deeper layers of the cornea. There may or may not be increased intra-ocular tension. In two patients studied by the author the disease began in one, a man, eighteen years after a successful cataract extraction, and in the other, a healthy woman, without apparent cause. All the typical symptoms recorded by Fuchs were present. Treatment is said to be unavailing, but in one of the author's patients the persistent use of dionin, associated with the internal administration of biniodid of mercury, seemed to check the process.

Filamentous Keratitis.—This somewhat unusual condition is characterized by the development of small threads or filaments of tissue on the cornea, which usually appear after abrasions or wounds (*traumatic filamentous keratitis*), or herpes, or occasionally without apparent cause (*spontaneous filamentous keratitis*). The tags have a bulbous extremity and are often twisted like a rope. They start from small vesicles by the formation of a slender pedicle, and are composed of epithelial cells, more or less degenerated, and sometimes especially elongated. Torsion of the filaments is due to the movement of the eyelids. A number of them may be found in a single cornea; thus, in a case reported by Zentmayer, fifteen to twenty of these filaments in various stages, some as clear vesicles attached to the cornea by a short pedicle, others as filaments 5 mm. in length with a bulbous extremity, were noted. They may speedily disappear, or persist, or recur after removal. Locally, holocain is of service.

Riband-like keratitis (*primary opacity of the cornea; transverse calcareous film of the cornea; keratitis trophica; keratitis petrificans* [Suker]) appears, as was pointed out by Nettleship, in two forms:

In the one, usually in elderly people, the exposed part of the cornea is invaded in a transverse direction by a *smooth subepithelial* opacity, oval in shape, which can be chipped off, and is composed of an incrustation of lime-salts. Hyalin deposits also appear in the cornea. There is no ulceration and no change in the overlying epithelium. The opacity is sharply limited and the remainder of the cornea is clear. The disorder almost invariably is symmetric, and is situated upon the exposed cornea, although deposits like the transverse band may also be found in other parts of the cornea. A margin of the cornea at each end is free. Gout and excess of uric acid in the blood have been suggested as constitutional causes, a suggestion strengthened by the occasional occurrence of insidious iritis, glaucoma, and hemorrhagic retinitis. It may be mistaken for the opacity which occurs from the injudicious use of salts of lead.

In the other type of the affection a horizontal band of opacity, grayish-brown in color, crosses the corneæ of eyes which have long been blind from iridocyclitis, sympathetic ophthalmia, and glaucoma. Here the stripe is less uniform, less sharply defined, and consists of a *roughened,*

transverse opacity. The calcareous nature of the other type may be wanting. As it occurs in the lower third of the cornea, or that part exposed when the eye is rolled up, and in an eye with impaired nutrition, the affection has been considered trophic in its nature. According to Best, the deposits are composed of lime and connective tissue. Hyalin globules are often present.

Blood-staining of the Cornea.—This phenomenon has been observed in cases of hyphemia and increased intra-ocular tension. The cornea assumes a smoky or rust-colored tint, except at its periphery, the clear portion being sharply separated from the cloudy area, which, however, is usually more pronounced in its center. The appearances closely resemble those of an amber-colored lens dislocated into the anterior chamber. With the microscope numerous granules (probably hematoidin) are found deposited in the substantia propria, which, according to Griffith, have entered the corneal tissues by endosmosis in a state of solution. The lesions have been studied by E. T. Collins, Vossius, Weeks, and J. Griffith; according to the observations of Collins and the author, it requires at least two years for the stains to disappear.

Arcus senilis (*gerontoxon*), or a circle of fatty degeneration of the substantia propria just within the margin of the cornea, is, as its name implies, almost invariably found in old persons. A true arcus is always separated from the adjacent sclera by a thin stripe of clear cornea. Occasionally a genuine example of this affection appears to have been noted in children (Hansell). An arciform opacity, the result of ulceration, may be mistaken for arcus senilis. The nature of the fatty material in arcus senilis has not been determined (Parsons).

Fig. 127.—Conical cornea.

The affection requires no treatment, and its presence appears not to interfere with the healing of wounds; for example, in cataract extraction.

Senile degeneration in the form of *sclerosis* and *atrophy of the corneal margin*, which, according to Fuchs, may arise in connection with arcus senilis, has been described on page 276.

Corneal pits (dellen) have been described by Fuchs, usually at the temporal margin of the cornea. They exist in the form of small saucer-like depressions, and may arise in association with swelling, inflammatory or otherwise, of the neighboring conjunctiva, or spontaneously. They last from a few hours to several days.

Conical Cornea (*Keratoconus*).—This consists of a cone-shaped bulging forward of the cornea, and is rarely congenital. It has been observed in several members of the same family. Usually stated to be more common in women than in men, some recent statistics do not confirm this assertion. It occurs most frequently between the ages of fifteen and thirty years; rarely it is seen in children. Its exact cause is unknown. Exhausting illness, menstrual disturbance, and especially chronic dyspepsia may be associated with the development of

conical cornea, the immediate cause being a disturbance in the relation of the intra-ocular pressure to the resistance of the cornea. Minute clefts in Descemet's membrane may appear, and pulsation of the cornea synchronous with each impulse of the heart. In the study of keratoconus Von Hippel has applied Abderhalden's test and found a disturbance of glandular secretion, an observation which has also been made by Siegrist.

The cone is transparent in most instances; occasionally its apex is slightly opaque. The bulging slowly progresses, but does not rupture or ulcerate. After years it comes to a standstill. One or both eyes may be involved, commonly the latter, the second eye being affected some time after its fellow. The eye becomes myopic and highly astigmatic. Slight forms of conical cornea may be overlooked unless the shadow-test is employed and the characteristic reflections observed, or a Placido disk is used (see page 50), or the cornea is studied with the ophthalmometer and the distortion of the images of the mires is investigated.

Treatment.—Although no form of glass or no optical apparatus may avail in advanced cases, a careful trial should always be made with spherocylindric lenses, and frequently their employment in unusual combinations will markedly improve visual acuteness. The refraction should be frequently investigated and the lenses changed according to existing conditions. The tendency for the cone to alter and increase in size seems to be lessened by the persistent use of eserin or pilocarpin. The strength of the solution need not be greater than $\frac{1}{12}$ to $\frac{1}{6}$ grain (0.0054–0.0108 gm.) to the ounce (30 c.c.). The administration of thyroid extract has been advised.

Properly perforated black disks—that is, forms of artificial iris—are recommended by L. Webster Fox for the relief of conical cornea.

If the apex of the cone appears to be thinning, a weak solution of sulphate of eserin and a compressing bandage are indicated.

In advanced cases an operation is advisable, having for its object the substitution of a contracting cicatrix for the tissue at the apex of the cone, which shall diminish the excessive curvature. Several plans are suggested: (1) Cutting off a small, superficial flap and subsequently cauterizing the surface, associated with repeated paracentesis of the cornea, and later a small iridectomy for optical purposes; (2) cutting off the flap and drawing the edges of the wound together with delicate sutures; (3) cutting from the apex of the cone a small disk with a trephine; (4) multiple punctures with fine needles; (5) obtaining the desired loss of substance by the application of a galvanocautery. If the resulting scar is directly central, an iridectomy for optical purposes will usually be required; but if the apex of the cone is eccentric, as it often is, iridectomy will not be required. Elschnig advises that the galvanocautery shall be applied at a dull-red heat to the apex of the cone, and this area connected with the nearest point of the corneal limbus by a superficially cauterized band. Optical iridectomy, according to this author, is not necessary.

Hydrophthalmos (*Hydrophthalmos congenitus; Keratoglobus; Megalocornea; Buphthalmos; Glaucoma congenitum*).—In this affection there is slow but progressive enlargement of the eye in all its diameters; the cornea is flattened, the pupil dilated and sluggish, the iris atrophic and sometimes tremulous, the sclera thinned and of a bluish color, and the anterior chamber deepened; the tension is raised. In the course of time the cornea may become cloudy (*keratoglobus turbidus*), although this is not always the case (*keratoglobus pellucidus*). The papilla may be deeply cupped. *Fissures in Descemet's membrane* may arise and appear as lines of grayish color with double contour, visible to the corneal microscope.

The affection appears at birth or shortly afterward, and its incipient stages are believed to be intra-uterine. The precise cause is not accurately determined. Buphthalmos is more frequent in negroes than in white children, perhaps, as Zentmayer suggests, on account of the greater frequency of congenital syphilis among them. It has been ascribed to an intra-uterine iridokeratitis with increased intra-ocular tension; in other words, a form of congenital glaucoma. Pyle divides the disease into two classes: true hydrophthalmos, depending upon congenital defective development of the cornea, iris, or filtration channels, and hydrophthalmos secondary to fetal intra-ocular inflammation.

The *prognosis* is unfavorable; the affection usually progresses to blindness. Iridectomy has been practised sometimes with a good result, more often with poor success; some favorable results from repeated sclerotomies have been reported; indeed, Haab states that if these procedures are begun early enough, infantile glaucoma can be cured. Corneoscleral trephining has been recommended; the author has observed one apparently beneficial effect from this procedure. Eserin or pilocarpin should be tried.

Injuries of the Cornea.—Traumatic Keratitis.—These comprise: (1) Foreign bodies; (2) erosions; (3) wounds, and (4) burns and scalds.

Foreign bodies, as particles of sand, cinders, fine splinters of iron, and bits of emery, may either lodge upon the epithelium or become embedded in the substance of the cornea. If they are sharp, like a splinter of iron or small thorn from a chestnut-burr, they may partially penetrate the membrane.

The pain of even a minute foreign body is considerable; the eye waters and grows red, and the source of irritation is commonly referred to the under surface of the upper lid, although the intruder may be directly upon the center of the cornea.

To remove an embedded foreign body a drop of a 4 per cent. solution of cocain or a 2 per cent. solution of holocain is instilled, the upper and lower lids are held apart with the thumb and forefinger of the surgeon's left hand, while with the right hand he takes a carefully sterilized needle, or a spud, and lifts the body from its position with as little injury as possible to the cornea. The area should afterward be inspected by means of a 2-inch lens and oblique illumination. In any case in which the operator is not sure that he has removed the foreign sub-

stance he may resort to the fluorescein method described on page 50. If the substance has been iron or emery, a small, rust-like spot will often remain. *Powder grains* may be removed by touching them with a fine galvanocautery point (E. Jackson).

If the spicule has partially penetrated, it may be necessary to pass a broad needle through the cornea behind it to secure a surface against which to work, and to prevent the manipulations from pushing it entirely through the cornea and into the anterior chamber.

After the removal of the foreign body, the resulting irritation may be allayed by a drop of atropin; the use of a bandage for a few days will facilitate the healing of the ulcer. Disinfection of the conjunctival cul de sac with a bichlorid lotion (1 : 8000) or one composed of cyanid of mercury (1 : 2000) is important. If in the attempt to remove the foreign body much abrasion of the cornea has occurred, great care should be exercised to prevent infection. Darier recommends a collyrium of cyanid of mercury which contains dionin.

Among oyster-shuckers a form of keratitis is prevalent (*oyster-shuckers' keratitis*), caused by small particles of oyster shells striking the cornea and producing ulcers. Randolph has shown that the disease depends upon the irritating chemical ingredients in the shell, and not upon micro-organisms. It is best treated by atropin and mild antiseptic lotions, and a 2 per cent. solution of holocain in physiologic salt solution. Kerato-iritis, the result of a bee-sting, has been reported (Huwald), and also from the action of antipyrin (Inouye).

Erosions.—A superficial loss of epithelium caused by the contact of a sharp body, for example, a finger-nail, in itself may be insignificant, but may lead, through septic infection, to a severe ulceration, particularly if the injured eye is exposed to the discharge from an inflamed lacrimonasal duct.

The *treatment* consists of the instillation of an antiseptic lotion, for example, bichlorid of mercury (1 : 8000), and the use of atropin and holocain, with a compressing bandage to immobilize the lids until healing takes place, provided no septic discharge is present.

Relapsing Traumatic Keratitis Bullosa (*Relapsing Erosion of the Cornea; Traumatic Keratalgia*).—In general terms the symptoms of this affection are these: Some time—several weeks or several months—after an abrasion of the cornea by a finger-nail, a twig, or similar object, the patient experiences, almost always on awakening in the morning, some difficulty in opening the eye, followed, when the lid is raised, by marked foreign-body sensation, decided epiphora, flushing of the eyeball, and sharp neuralgic pain. Each movement of the lid is painful, and the "attack" continues from one-half hour to several hours, when, usually by afternoon, the symptoms subside and the eye is again apparently normal. Careful examination during the continuance of the irritative signs just described will reveal on the cornea a small ruptured vesicle, or a larger blister or bulla, or sometimes simply an erosion of the superficial epithelium, without indications of vesicle or bulla. Occasionally the only lesion to be detected is the scar or macula caused by the original

injury, without loss of epithelium. These attacks may recur at short or long intervals, for weeks, months, and even years.

Treatment.—The ordinary treatment of corneal ulcer is indicated, and the author has been especially satisfied with the action of holocain (2 per cent.), persistently and frequently used. A pressure bandage and massage with a salve of yellow oxid of mercury may be tried. A drop of liquid vaselin instilled at bedtime is useful.

Wounds of the cornea naturally divide themselves into *non-penetrating* and *penetrating*, and differ in character according to the instrument which has inflicted them.

Non-penetrating wounds partake of the nature of erosions, and, like them, may be in themselves of minor importance, but may result in sloughing ulcers through microbic infection.

The treatment already described is applicable.

A *penetrating* wound allows the escape of the aqueous and renders incarceration and prolapse of the iris liable, with all the possibilities described in connection with perforating ulcers. The wound may injure the lens and cause traumatic cataract, or involve the ciliary region and cause sympathetic inflammation, or become infected and originate a sloughing keratitis or a panophthalmitis.

After a perforating wound of the cornea the eye should be thoroughly disinfected, the iris, if prolapsed, replaced if possible, and eserin or atropin instilled, according to the situation of the injury. If replacement is not possible, and usually it is not, the prolapsed portion should be seized with iris forceps and excised. In either event the subsequent treatment requires rest, disinfection of the conjunctival cul de sac, and a carefully applied antiseptic compressing bandage. Care must be taken to ascertain whether a foreign body has lodged within the anterior chamber or within the deeper portion of the globe. *x*-Ray examination may be necessary.

The tendency to *traumatic iritis* may be combated by the frequent use of cold compresses, and the instillation of an atropin solution. In severe corneal wounds, involving the iris, lens, and ciliary body, the question of enucleation or evisceration must be decided. Gaping wounds of the cornea may be closed with fine silk sutures or, according to de Wecker, covered with conjunctiva, which is dissected loose in such a manner that it may be united over the cornea by a purse-string suture. After the corneal wound has united, the conjunctival covering is removed and restored to its original position. Kuhnt's conjunctival flaps may also be used.

Burns and **scalds** are produced by the contact of acids, lime, molten metal, and hot water or steam, and the general management of such cases does not differ from that of similar accidents to the conjunctiva, which necessarily is involved (see page 239).

Sometimes the burn may be superficial and the whole surface epithelium be changed into a white scum, which presents a most alarming appearance. The destroyed tissue, however, is speedily replaced by a new layer of epithelium. Burns with slaking lime and molten metal

are liable to result in disastrous consequences, and may be followed by sloughing keratitis and even panophthalmitis, and if the burns are located at the limbus, elevation and intra-ocular pressure may develop (Kümmell). Allport and Rochester have advantageously used intramuscular injections of cacodylate of sodium in the treatment of corneal opacity following a lime burn. *Ammonia burns* of the eye are of serious import, and even where the injury originally seems to be comparatively slight, there may develop later rapid necrosis of the cornea, with exudation in the anterior chamber, followed by blindness. There is a certain similarity between *carbolic acid burns* and those caused by ammonia, but the ultimate prognosis is, according to Stieren, less gloomy.

Severe corneal and conjunctival burns have been caused by *exploding golf-balls*, so-called "zodiac" and "water-core" balls, containing caustic contents, *e. g.*, barium sulphate, sodium hydrate, soap, free alkali, and zinc chlorid. The *treatment* of burns and scalds has already been described (see page 239).

All the various forms of corneal injury cause more or less severe inflammation, properly classed under the general term *traumatic keratitis*, and possesses in greater or less degree the cardinal symptoms of keratitis—pain, lacrimation, photophobia, and disturbance of vision.

Peripheral Annular Infiltration of the Cornea (*Ring Abscess of the Cornea*).—This condition is characterized by an infiltration of the cornea, the exudation being distributed in a zone concentric with the corneal margin. At first the ring is gray, but rapidly becomes yellow, its inner edges being somewhat less well defined than its outer; in almost all cases panophthalmitis is the ultimate result. The condition most commonly follows perforating wounds of the cornea, especially if caused by chips of metal, and operations, for example, cataract extraction. Rarely is it seen after perforating corneal ulcers and in metastatic ophthalmitis. It has been well studied by Fuchs, and recently by Morax. Bacteria enter the anterior chamber through a wound and there proliferate, and by their products give rise to an infected iridocyclitis and keratitis, by which the cornea is attacked from the rear. Leukocytic infiltration in the form of a ring follows, which is itself amicrobic. Hanke believes he has found the specific bacillus, but Morax maintains that so-called ring abscess cannot be explained by the presence of any one specially determined microbe, but by the proliferation of certain microbes in the anterior chamber, among which he is willing to admit Hanke's bacillus.

Traumatic Striped Keratitis (*Keratitis Striata*).—This condition may arise after incised wounds of the cornea, and in its most perfect manifestation, after cataract extraction (see page 667), and especially after expression of the lens in its capsule (see page 660). The gray striæ, which should be studied with a loupe, are disposed perpendicularly to the wound, and stretch toward the opposite margin of the cornea. They cause no irritation, and the appearance does not materially complicate the treatment of the wound which gives rise to them. These

stripes do not represent a cellular infiltration, but depend upon folds in Descemet's membrane. Usually they disappear within a week of their development.

Obstetric Injuries of the Cornea.—These injuries may be due to prolonged labor or to forceps pressure. They have been particularly well studied by Ernest Thomson and Leslie Buchanan, who classify them as diffuse temporary opacities due to edema, and permanent linear opacities, which extend vertically, obliquely, or horizontally across a whole or a part of the cornea, and which are caused by rupture of the posterior elastic lamina of the cornea. In several cases of the edematous variety studied by the author the opacity gradually but entirely disappeared. Doubtless some of the scars of the cornea, known as "congenital leukomas," have been caused by birth injuries. The author has investigated a few cases of high unilateral irregular corneal astigmatism evidently due to the same cause.

Fig. 128.—Dermoid of the cornea (from a patient in the Philadelphia General Hospital).

Fig. 129.—Dermoid of the corneo-scleral junction, which on section showed a gland inclusion (from a patient in the University Hospital).

Tumors and Cysts of the Cornea.—Tumors of the cornea are rare. The following have been described: fibroma, papilloma, myxoma, dermoids, sarcoma, epithelioma, and endothelioma. True fibromas have been reported, but usually they are *scar-fibromas*, that is, they are hyperplastic scars. A myxomatous degeneration of such a scar is the probable origin of the so-called corneal myxomas. The papillomas, which have been examined, in most instances have arisen at the limbus and invaded the cornea. A few primary sarcomas, even in children, have been described, and Parsons has investigated one endothelioma. According to this authority, nearly all of the cases of so-called epithelioma of the cornea are growths beginning in the limbus at the position where the conjunctival changes into corneal epithelium. This was the condition of affairs in several specimens examined by the author.

Dermoid tumor is a congenital growth, and sometimes is associated with other anomalies of the lid and eyes. Strictly limited to the cornea, it is most uncommon; generally it occurs as a firm, hemispheric, yellowish-white growth, lying partly upon the cornea and partly upon the

conjunctiva. The apex, often paler than the rest of the growth, is covered with short hairs. These, however, occasionally grow to an unusual length, and have been seen protruding through the fissure of the lids and hanging down upon the cheeks. If undisturbed, the tumor may slowly enlarge, and has been reported to have attained the size of a walnut. Bilateral dermoids have been recorded.

These dermoids have been ascribed by Van Duyse to the remains of amniotic adhesions, and by Remak to invagination of the ectoderm. Microscopically, the growth represents the structure of the skin and its appendages.

Teratoid tumors may be situated at the corneoscleral junction, rarely, if ever, on the cornea, or upon the outer half of the sclera, or upon the bulbus in the neighborhood of the caruncle. They contain, as a rule, acinotubular glands, fatty tissue, smooth and striated muscle-fiber, hyalin bodies, and cartilage.

Corneal cysts, according to Oatman, occur in two principal forms—*epithelial* and *lymphatic*. The first variety is the more common; it usually arises from the epithelial layer of the conjunctiva, which is ingrafted on the cornea. Cysts following injury of the cornea have been ascribed to the proliferation of surface epithelium which has been carried into the corneal stroma. They are, therefore, *implantation cysts*. Oatman doubted this pathogenesis, and explained them by assuming that a proliferation of superficial epithelium lines the wound with epithelial cells, and the mass thus produced, separated from connections with the surface, takes on an active growth and forms the cyst. *True lymphatic retention cysts* may result from dilatation of the corneal canals and spaces. Corneal cysts may be cured by excising a piece of their walls.

Congenital Anomalies of the Cornea.—*Microphthalmos* is that condition in which the entire eye remains in a more or less rudimentary state, and in which the cornea is too small in all its diameters. Pure cases of microphthalmos, according to Manz, are very rare; usually one or other of the component portions of the globe is wanting. Numerous theories have been advanced to explain their etiology—incomplete closure of the fetal ocular cleft (Arlt), fetal illness *in orbita* (Wedl and Boch), intra-uterine sclerochorioretinitis (Deutschmann). The affection has also been ascribed to the influence of heredity.

Megalophthalmos has been described on page 280.

Sclerophthalmia or *sclerosis* is that condition in which the opacity of the sclerotic encroaches upon the cornea in such a manner that only the central portion remains transparent. It is due to an imperfect differentiation of the cornea and sclera at an early period of fetal life. It may be symmetric, and affect only the upper half of the cornea.

Congenital opacities of the cornea are seen in the form of milky spots which may clear up in later life, or as leukomas. Usually the iris is dimly visible through the clouded tissue. These opacities are due either to intra-uterine inflammation or to an arrest of development (see also Birth Injuries, page 284).

Congenital anterior staphyloma of the cornea appears in the form of a true staphyloma, and is a rare affection. The abnormality depends not so much upon a malformation or an arrest of development as upon a fetal inflammation, which, according to Pincus, takes place in the second half of fetal life. They have been well studied by J. Herbert Parsons, who holds that the lesions develop in exactly the same manner as they do when they take place after birth. Treacher Collins, however, thinks failure of the development of the anterior chamber may be the original cause in some cases. Peters ascribes the condition to defective development of Descemet's membrane, and Von Hippel to *internal ulcer of the cornea.* Heredity probably plays some rôle in this and similar affections of the cornea. Congenital staphyloma of the cornea associated with dermoid formation has been reported.

Congenital melanosis or *pigmentation of the cornea* may appear in the form of a vertically oval area of brownish color in the center of this membrane. The affection is more common in women than in men. It has been ascribed to an abnormal development of the uveal tract (Krukenberg). The lesion has been well studied in this country by Dr. T. B. Holloway, and he has described *peripheral pigmentation of the cornea* in association with symptoms suggesting multiple sclerosis; some of the patients have cirrhosis of the liver. Congenital familial flatness of the cornea (*cornea plana*), according to Ruel, is characterized by flattening of the anterior portion of the eyeball in such a manner that the curvature of the cornea passes directly into the curve of the sclera without the formation of an angle. In Ruel's patients the corneas were diffusely opaque.

CHAPTER VIII

DISEASES OF THE SCLERA

The sclera, constituting four-fifths of the covering of the globe of the eye, and being in intimate relationship with the choroid and ciliary body, is subject to inflammations peculiar to itself, and to changes indicative of disease of these subjacent structures. Its close connection with the cornea associates the latter membrane in some phases of its diseases, and its union with the iris through the pectinate ligament establishes an anatomic connection, just as there often is a pathologic relation. The overlying bulbar conjunctiva necessarily participates in scleral inflammation.

The inflammations affect (1) the episcleral tissue (*episcleritis*) and (2) the sclera itself (*scleritis*), and hence are *superficial* or *deep*. They further are *acute* or *chronic*, *diffuse* or *circumscribed*.

Episcleritis occurs in the form of small, dusky red, subconjunctival swellings or nodes, which usually appear in the ciliary region on the temporal side of the cornea, though patches may occur in any portion of the zone.

The conjunctival vessels over the patch are coarsely injected, and movable with the somewhat edematous conjunctiva. The episcleral vessels show a dusky congestion which is immovable. The elevation is back shaped; sometimes tender to pressure and sometimes not, and there may or may not be much irritation and pain. In some cases of phlyctenular disease of the corneal margin it is difficult to decide between this affection and episcleritis; what appears to be a patch of the latter may develop into the former.

The disease runs a subacute course, reaching its height in about three weeks, then gradually disappears, and leaves a somewhat dull area of discoloration marking its former position. Relapses are frequent, both at the original seat or in new spots on the sclera, and these recurrences may happen again and again for months and even years. The cornea and uveal tract easily participate in the inflammation.

Cause.—Episcleritis and *episcleral nodes*, either solid or moderately soft in texture, are more frequent in women than in men in the author's experience. Patches of episcleritis of the character described occur in the eyes of those who are much exposed to the weather. In other cases superficial scleritis is caused by rheumatism, gout, tuberculosis, menstrual derangements, and enterogenous auto-intoxication, and also appears without discoverable cause. Well-marked episcleral (not conjunctival) congestion or episcleritis occurs in connection with disease of the accessory sinuses.

In these forms of superficial scleritis the *prognosis* is good so far as sight is concerned, because deeper and adjacent structures are uninvolved, but unfavorable on account of the recurrences.

Treatment.—This consists in the use of atropin to allay pain and prevent any tendency to iritis, warm antiseptic collyria, and hot compresses. Dionin is of distinct service. In the chronic types eserin and pilocarpin, $\frac{1}{4}$ to $\frac{1}{2}$ grain (0.0162–0.324 gm.) to the ounce (30 c.c.) of water, have a beneficial influence, provided no iritis is present. Subconjunctival injections of salt solution are useful, and similar injections of salicylate of sodium (2 per cent.) and of hetol (cinnamate of sodium) have been recommended (Pflüger). Massage with a salve of the yellow oxid of mercury is indicated in chronic cases, and it has been recommended to scarify the tumefaction, scrape it away with a sharp curet, or cauterize it repeatedly, in a superficial manner, with the actual cautery. These cauterizations are often followed by excellent results. Internally, salicylic acid and iodid of potassium are needed in rheumatic cases, and good results follow diaphoresis with pilocarpin or the Turkish bath. Menstrual and uterine disorders must be rectified, and the influence of intestinal sepsis eliminated. If a tuberculous taint is discovered, injections of *tuberculin* are of service. Any error of refraction or anomaly of the external eye muscles should be corrected.

Fugacious Periodic Episcleritis.—This name has recently been applied by Fuchs to a variety of relapsing episcleritis characterized by the appearance of one or more patches of episcleral injection or edema, of violaceous hue, lasting from two to eight days, and reappearing again at intervals of several weeks or even months, to go through the same course. The duration of the affection is usually about one year; it occurs most frequently in adults. Gout and rheumatism are associated constitutional conditions. The same affection was described some years ago by Swan M. Burnett under the name of "Vasomotor Dilatation of the Vessels," and by Jonathan Hutchinson with the title "Hot Eye." The treatment is the same as that already recommended for episcleritis.

Scleritis may appear in the form of a *diffuse,* bluish-red injection, occupying the entire exposed portion of the sclera, very painful, unattended with secretion, save some increase in lacrimation, and liable to be mistaken for conjunctivitis or iritis; or in the form of *circumscribed* patches, of violaceous tint, situated in the ciliary region, and somewhat resembling in appearance the forms of superficial or episcleral elevations just described, being, however, less sharply defined, so that the whole zone may be involved, but in unequal degree. Spicer calls attention to crescentic areas of infiltration in the cornea, separated from the patch of scleral inflammation by a band of clear cornea. In many cases of diffuse deep scleritis, hard, whitish nodules develop in the inflamed tissue (*nodular scleritis*). Overgrowth of the infiltrated tissue may produce diffuse or circumscribed areas, which are called *hyperplastic scleritis.* The chief distinction between the *superficial* and *deep* forms

of scleral inflammation is the almost invariable tendency of the latter to affect other portions of the eye—the cornea and uveal tract.

Pathology.—In episcleritis the infiltrating cells are found either in the superficial layers around the conjunctival vessels or in the deepest layers. The vessels are dilated, extravasations of blood are found, and often spots of necrosis and giant cells. Usually the choroid and sclera are infiltrated and edematous.

The **causes** of deep scleritis are exposure to cold, rheumatism, gout, scrofula, vasomotor changes, and disturbances of the sexual apparatus, especially anomalies of menstruation. Young adults are most frequently attacked. The so-called *gummatous scleritis*, in which the patches are yellowish brown and translucent, is due to syphilis; and gonorrhea, if it is associated with synovitis, may cause the disorder. Deep scleritis is also seen in the subjects of congenital syphilis and tuberculosis. The trial of tuberculin in scleritis will often be followed by a general as well as a local reaction. Excised nodules may show epithelioid cells and giant cells, but no tubercle bacilli. *Tuberculous scleritis* is probably due to an infection derived from the aqueous arising from the filtration angle (Verhoeff). Finally, types of scleritis (sclerokeratitis) unassociated with any definite cause or diathesis are seen in young and middle-aged subjects, most commonly women, whose nutrition is depressed, and who may or may not have a tuberculous disposition or inheritance. Scleritis may be a metastatic inflammation, the original focus of infection being at some distant part of the body, for example, a rectal abscess, a felon, or a purulent sinusitis (Dupuy-Dutemps).

Fig. 130.—Tuberculous sclerokeratitis, showing scleral nodules and characteristic triangular corneal infiltration (from a patient in the University Hospital).

Deep scleritis usually attacks both eyes, runs a chronic course, and may affect the iris (leading to closure of the pupil), ciliary body, choroid, vitreous (causing opacities), and the cornea. In prolonged cases of the disease dark scars remain after absorption of the products of the inflammation, which are unable to resist the intra-ocular pressure, and form elevations (ectasia scleræ). Sometimes the whole anterior portion of the sclera becomes bluish or slaty colored, is misshapen and elongated, and the cornea, which appears small, is poorly differentiated from it on account of the haziness of its margins.

Sclerokerato-iritis (*Scrofulous Scleritis; Anterior Choroiditis*).—This name is applied to the complicated scleritis referred to in the previous paragraph, and is characterized by chronicity, relapses, and involvement of the cornea and iris.

Beginning with a deep scleritis of the ciliary zone, the adjacent cornea becomes opaque and sometimes ulcerates; the iris is inflamed, posterior synechiæ form, and pain and congestion may be severe. After weeks the symptoms subside, the characteristic discolored area marks the former scleral disease, and haziness in the cornea indicates the seat of previous inflammation in this membrane. Relapse takes place, with fresh scleritis, new corneal involvement, renewed iritis, or iridochoroiditis, and vitreous changes, and so on, until after many months, it may be, the disease comes to an end, leaving the sclera discolored and bulged, the cornea covered with patch-like opacities, the iris bound down with adhesions, the vitreous filled with opacities, and the eye practically deprived of vision.

Sclerotizing keratitis, referred to on page 274, is the name applied to a patch of opacity in the deeper corneal layers, usually triangular in shape, with its base toward the patch of scleritis, which is its origin. After the cure of the scleritis, a white or yellowish-white opacity remains directly in contact with the sclera. Instead of a single patch of this character, several small triangular areas may arise in the circumference of the cornea as the result of scleritis.

Treatment.—The treatment of scleritis and sclerokerato-iritis depends upon the type and stage of the disease and the presence or absence of definite cause. Locally, atropin, hot compresses, cocain, holocain, dionin, and boric acid lotion, and in painful cases leeches to the temple are suitable. Pilocarpin is valuable if iritis is not present. The eyes should be carefully protected with goggles. After the subsidence of acute symptoms massage may be tried. The use of the actual cautery has been mentioned. Subconjunctival saline injections are useful.

In rheumatic cases salol, the salicylates, the alkalis, and iodid of potassium are the most available remedies; in gout, carefully regulated diet, mineral waters,—Buffalo, Poland, etc.,—citrate of lithium, and colchicum, especially in the form of colchicin, and change of climate are useful. Cod-liver oil, iodin, iron, and sweats with pilocarpin, $\frac{1}{10}$ grain (0.00648 gm.) hypodermically, are also indicated. The diaphoretic measures are proper in any case, other things being equal, and, in place of pilocarpin, an electric cabinet or Turkish bath may be the means of creating diaphoresis. In syphilis, bichlorid of mercury, inunctions of mercurial ointment, and neosalvarsan are efficacious. Indeed, mercury is generally advantageous as a means of altering the nutrition of the part and preventing exudation into the uveal tract. Disorders of menstruation should always be corrected. Finally, in subjects with depressed nutrition, quinin, arsenic, and a general tonic regimen are required. Because many cases of scleritis are due to tuberculosis a tuberculin treatment (see page 318) is often indi-

cated, and may be followed by most satisfactory results. Tests with tuberculin should always be made; a positive reaction will follow in a number of instances.

Annular Scleritis (*Brawny Infiltration of the Sclera*).—To this severe form of scleritis, which invariably affects the whole region around the cornea, J. Herbert Parsons, G. Derby, and Verhoeff have recently called renewed attention. Unlike ordinary scleritis, which usually attacks young adults, this affection is a disease of advanced age or, at least, of middle life. Both eyes are usually affected, though not to an equal extent. The disease is essentially chronic, and subject to periodic exacerbations and remissions. Verhoeff believes that syphilis is an important etiologic factor, although this cause is doubted by Schodtmann, who first described the disease. Gilbert has ascribed it to gout. The prognosis is most unfavorable, many of the eyes having been lost. The corneal margin is the essential site of the infiltration, from which region it spreads on both sides into the surrounding tissues, overlapping the cornea on the one side and extending as far as the equator of the eyeball posteriorly on the other. The swelling is usually gelatinous and succulent and has a brownish-red color. In addition to the involvement of the cornea, the uveal tract, especially the anterior part of the choroid and the ciliary body, are inflamed.

Posterior Scleritis.—In this affection, as described by Fuchs, the symptoms are edema of the lid, exophthalmos, conjunctival chemosis, and ophthalmoscopically the appearance of detachment of the retina, or of a gray cloudiness over the affected area. After the subsidence of the inflammation changes in the retina remain. Secondary iridocyclitis may arise. According to Coats, the disease depends upon blocking of one of the larger ciliary arteries and consequent infarction of the inner layers of the sclera, choroid, and retina.

Staphyloma of the sclera has been divided by systematic writers into *anterior*, *equatorial*, and *posterior* staphyloma, according to the situation of the lesion. Posterior staphyloma is detected with the ophthalmoscope in a highly myopic eye (see page 131).

It is evident that all bulging of the sclera depends upon a disturbance between the resistance of the sclera and the intra-ocular tension, but it is not evident in all cases whether the process which originated the trouble began in the underlying tissue or in the scleral structure itself. There may be a general enlargement of the scleral coat, for example, in hydrophthalmos (see page 280); or one or more darkly tinted swellings in the ciliary region may arise, one sometimes occurring in advance of each rectus tendon; or, finally, the staphylomatous swelling may exist at the equator in the region of the vena vorticosa.

The following **causes** may originate scleral staphyloma: Chronic glaucoma, old kerato-iritis and closure of the pupil, inflammation of the ciliary body, thinning of the scleral coat by repeated attacks of inflammation, tumors, and wounds closed by non-resisting scars.

Treatment.—A single scleral staphyloma may not destroy vision. If the intra-ocular tension is increased, an iridectomy is indicated.

If the eye is useless, enucleation or one of its substitutes may be necessary.

Abscess and **ulcers of the sclera** are exceedingly uncommon. Abscess in the scleral tissue may result from an infected wound and has been seen in connection with certain specific and contagious diseases—*e. g.*, glanders. Metastatic scleral infection has been recorded (see page 289).

Ulcer of the episcleral tissue has been described in association with tuberculosis. A tumor, gumma, or tubercle of another region of the eye may break down and ulcerate into the sclera.

Tumors of the sclera are rare growths. The following have been described: Fibroma, fibrochondroma, enchondroma, and osteoma.

Primary sarcoma, if it exists, must be rare; recently a case has been recorded; secondary sarcoma, carcinoma, and glioma have been reported. Gumma of the sclera has been described, and tubercle may invade it from the uveal tract. A few *scleral cysts* have been recorded. Small primary scleral growths may be dissected from their beds, and the wounds closed with conjunctival sutures (Fig. 131).

Fig. 131.—Cyst of the corneoscleral junction (Philadelphia General Hospital).

Injuries of the Sclera.—Wounds of the sclera may be caused by a sharp implement (knife, scissors, broken glass, etc.) or foreign body (chip of iron or steel, bullet, etc.), or they may result from a blow on the bulbus on the inner side and above, rarely downward and out, causing *rupture of the sclera*, usually found 3 mm. from, and concentric with, the corneal margin (T. Collins). The rupture may be exposed through a rent in the conjunctiva, and is then said to be "compound," or it may be concealed by the conjunctiva, which is untorn. Indirect ruptures of the sclera occur in the vicinity of the cornea and are more or less concentric with it, because here, due to the presence of Schlemm's canal and the penetrating anterior ciliary veins, there is an area of poor resistance. A blow may also *rupture the cornea*. Corneal tears, according to L. Müller, are more common in young people than scleral ruptures.

If the wound has *perforated* the sclera, two complications are liable to be present: loss of a portion of the contents of the globe and injury to the inner coats, and the introduction into the eye of septic material which will cause destructive inflammation.

Symptoms.—A perforating wound of the sclera, if sufficiently large, causes loss in the tension of the globe, hemorrhage into the vitreous, or, it may be, into the anterior chamber, and the appearance of dark tissue in the wound, representing, according to its situation, portions of the

choroid or ciliary body; a bead of vitreous is likely to present. A small perforating scleral wound may be hidden by the overlying contused and swollen conjunctiva. Usually the intra-ocular tension is lower than normal. Rupture of the sclera is commonly associated with grave lesions in other portions of the eye—separation of the retina and extensive tears in the choroid and iris.

Prognosis.—This depends upon (1) the extent and situation of the wound and amount of escape of vitreous; (2) the presence or absence of septic material upon the implement or body which inflicted the injury; (3) whether a foreign body has remained within the globe; and (4) the character of the foreign body which may have entered. It is evident that even a trifling perforating wound, unattended with loss of vitreous or prolapse of the inner coats, may be a point of entrance of infection.

Treatment.—Having carefully ascertained that no foreign body is within the globe, the eye should be disinfected with a solution of bichlorid of mercury (1 : 5000), and the edges of the wound, after all foreign substances have been removed, penciled with a stronger solution of the same drug (1 : 2000) or with a 5 per cent. solution of iodin. The overlying conjunctiva is next drawn together with several fine sutures. The eye is closed with an antiseptic compressing bandage and the patient is put to bed. Iced compresses are an advantage during the early stages of the treatment. At the end of forty-eight hours the wound may be inspected and the dressings renewed. In larger wounds the sutures (sterile silk or catgut) are passed directly through the sclera by some surgeons, care being taken to avoid the choroid, but the author agrees with Snell that usually scleral sutures are not necessary, conjunctival sutures being sufficient; the sutures may be removed at the end of a week if the healing has progressed favorably. Some surgeons advise the introduction of iodoform before the application of the bandage. In some instances, in spite of kind healing of the scleral wound, there are subsequent detachment of the retina, vitreous change, and shrinking of the eyeball, but apparently hopelessly injured eyes may be saved by careful aseptic surgery.

In the event of a scleral wound being extensive, with much loss of vitreous and collapse of the coats, especially if the ciliary body is involved and sight practically gone, or if the endeavors to remove the foreign body have been unsuccessful, enucleation should be performed to avoid the dangers of sympathetic inflammation in the fellow eye.

Foreign Bodies.—If the wounding substance has been small—*e. g.*, a chip of steel, a splinter of glass, a particle of dynamite cap, or a bullet—endeavor should be made to ascertain whether this has penetrated the globe and remained within it, or has passed entirely through the eyeball and buried itself in the tissues of the orbit. Foreign bodies may be embedded in any of the structures of the eye and are frequently found in the vitreous. If loose, they tend to gravitate to the lowest part of the vitreous and rest upon the posterior part of the ciliary body (T. Collins). *Double perforation* of the eye is not uncommon as the result of a

bullet wound or one caused by the explosion of a dynamite cap, but less frequent if the foreign body is a chip of iron or steel. In rare instances the perforation of the posterior scleral wall has been discovered with the ophthalmoscope, but since the introduction of *x*-ray examination the diagnosis is rendered comparatively easy, and the radiographs should show whether the foreign body has passed entirely through the posterior scleral wall or is embedded partly within and partly without the scleral covering. According to Leber, perforating injuries of the eye with pieces of copper may result in purulent inflammation merely by the chemical action of the metal; if microbes are proved to be absent by culture experiments, an attempt to remove the body may be made, and, if successful, the eye saved, even if inflammation has begun. Foreign bodies may be tolerated for long periods of time, with good vision, in the background of the eye, but can never be trusted unless they are small and the accompanying lesions trifling; in other circumstances they are liable to cause degenerative changes. However, small fragments of glass have remained for years within the globe without creating untoward symptoms.

Unfortunately, blood in the vitreous and anterior chamber, or opacity of the lens, is apt to obscure the media to such a degree that ophthalmoscopic examination is not of much service; but if the media are clear, this method may be the means of detecting the foreign body. Air bubbles in the vitreous are suggestive, but not pathognomonic of a foreign body in the globe. An attempt at locating the body may be made by observing the situation of the wound, the condition of the capsule of the lens, the probable direction which the foreign substance took on making its entrance, by a search for points of tenderness and for a scotoma in the field of vision. If there is any doubt, a skiagraphic examination should be undertaken, and in the majority of instances (pieces of wood excepted) the Röntgen rays will readily reveal the presence and position of the foreign body. Of the various methods devised for this purpose, the one elaborated by W. M. Sweet, in the opinion of the author, is most satisfactory. The method of McKenzie Davidson and Dixon's modification of the Sweet method are also excellent.

Having satisfied himself of the presence and position of a nonmetallic foreign body within the globe, the surgeon may attempt to extract it through the original wound with delicate, carefully disinfected forceps, or through a new wound made in the most favorable situation. But in some instances a small fragment of sterile glass, for example, difficult or almost impossible to reach had better be allowed to remain rather than to make matters worse by the frequent introduction of instruments into the vitreous chamber in the effort to extract it.

If the foreign body is composed of iron or steel and its presence cannot be detected on account of opacities in the media, a diagnosis may be made, as was first suggested by T. R. Pooley, with the magnetic needle. Useful instruments have been constructed on this principle by Asmus and Hirschberg, and are known as *sideroscopes*, with which a

properly protected magnetic needle is brought near different portions of the eye in succession, and any deviation of the needle carefully noted. Where the deviation is greatest there is reason to suspect the foreign body exists. A large magnet may also be employed for diagnostic purposes, the dislodgment of the foreign particle giving rise to a localized spot of pain or point of bulging, but great care must be exercised in attempting this procedure, as sudden movement of the foreign body may cause serious intra-ocular lesions, hemorrhage, tearing of the iris, etc. Moreover, as has been pointed out by Hirschberg, the absence of pain when the eye is approached by the magnet does not surely exclude the presence of a foreign body.

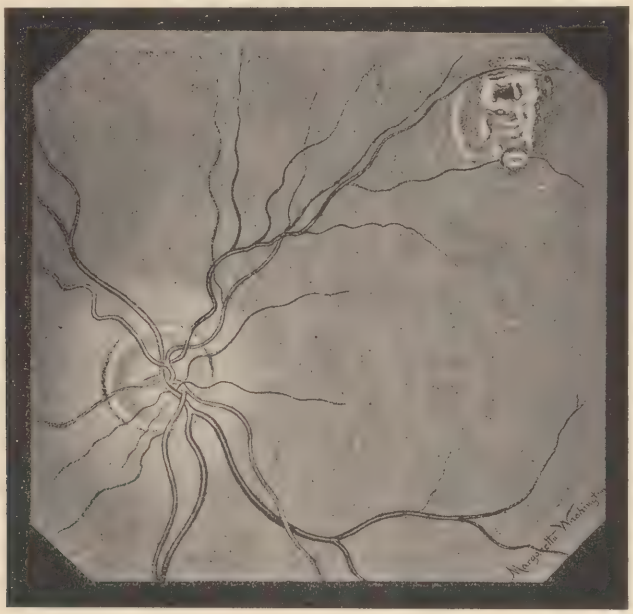


Fig. 132.—Foreign body in the vitreous.

The most satisfactory method in a doubtful case, and where the media are so obscured that ophthalmoscopic examination is impossible, is the employment of the *x*-rays, which, if properly used according to Sweet's method (see Appendix, page 692), have, in the author's experience, never failed to give exact information of the position of the foreign substance. When this has been determined, the body should be extracted with the electromagnet. In former times it was much the practice to introduce the extension point of a magnet, for example, the Hirschberg model, either through the original entrance-wound or through one made for that purpose, as far into the vitreous as was necessary to attract the splinter of iron or steel from its position. Naturally, the disadvantage of this procedure is the introduction of an instrument into the vitreous chamber, and hence the value of the so-called *giant*

magnets, notable among which is the Haab model. If, however, a perfectly accurate localization has been made, for example, by the Sweet method, there is usually very little difficulty in making a very small incision with a Graefe knife through the sclera at that point which nearest approaches to the position occupied by the particle of metal. To the lips of this wound the extension point of a magnet of sufficient power may be applied, which almost invariably attracts the body to its point. It is to be noted that the instrument is not introduced within the vitreous, but placed against the lips of the wound, which afterward is closed in the usual manner and the treatment already recommended in connection with scleral wounds is instituted. Instead of this procedure, the various giant magnets may be employed to draw the body from its position in the posterior portion of the eye, around the lens into the anterior chamber, from which it is readily removed by a small hand magnet through a suitably placed corneal incision. This is the method especially advocated by Haab, and which is fully described on page 642. Of the great value of this method there can be no doubt, especially if it is not possible to utilize the *x*-rays for diagnostic purposes, in which circumstance it is the only proper procedure, but if the foreign body can be accurately localized by the Röntgen rays, according to Sweet's method or any other satisfactory method, and the position of the body found to be such that a small incision may be made directly over it, or in its immediate neighborhood, through which it is drawn by a magnet (the Sweet model has proved most satisfactory), without the introduction of the instrument into the vitreous, the results are just as good as those which follow the withdrawal of the foreign body by means of a giant magnet. Direct extraction in these circumstances presents no greater danger than the other method, and in many instances a danger not so great, and such accidents (and there are now many of them on record) of intra-ocular hemorrhage, sudden tearing of surrounding tissues, especially of the iris, and the like, are avoided. Haab, however, strongly condemns the extraction of steel or iron from the interior of the eye through a scleral incision, and his opinion in this respect is shared by many surgeons. Thus, Charles Goulden examined the records of 118 extractions of metallic foreign bodies from the eye, and found that the worst ultimate results occurred if the body entered through the sclera or if it was removed by scleral puncture. The author's experience, on the other hand, with direct extraction of magnetic particles in the manner described after *x*-ray localization, has been most favorable. Modifications of the Haab magnet, less powerful, but also less bulky, have been devised by Lippincott, Johnson, Sweet, Parker, and others. Other giant magnets are those, for example, devised by Volkmann and Hirschberg, and Mellinger has designed the *Innenpol magnet*, in which the patient's head is placed within a large electric coil, and the passage of the current magnetizes both the foreign body in the eye and the iron instrument held in front of the eye to attract it.

If infection has already begun when the patient is seen, and the

condition of the eye is not so hopeless that immediate enucleation is necessary, various methods have been tried to check the purulent process. If, for example, the wound of entrance has been through the cornea, and the anterior chamber contains pus, this may be evacuated, and, as Haab recommends, small rods of sterilized iodoform, one or two, according to circumstances, may be introduced within this chamber. He also, in like manner, introduces these rods directly into the vitreous if infection has begun in that region, and reports successes. This method of intra-ocular disinfection has been sharply criticised—Krause, for example, believing that iodoform, either in the form of powder or rods, is unable to influence favorably beginning infection, but that, on the contrary, of itself it may produce pathologic changes, and represents a method inferior to other well-known procedures for the relief of infected wounds—for example, that of Schirmer, who brings the patient under the influence of mercury by inunctions. The author has tried the iodoform method in several cases with indifferent success, and prefers the use of mercury, especially calomel, in repeated, properly guarded, doses. Drainage of the anterior chamber, expression of the lens, and thorough irrigation of the posterior chamber with salt solution have seemed to be of service in a few cases in the author's practice. Constant iced compresses are of value. Van Millingen has suggested the trial of endocular cauterization in these circumstances—that is, the introduction into an infected scleral wound of a galvanocautery point, if necessary, even into the vitreous, and the cauterization of all surrounding tissue.

Prognosis.—This is always grave, but by means of the methods just detailed many eyes have been saved, and some with useful vision. The important point is to operate as soon as possible after the accident —*i. e.*, before the foreign substance has become incarcerated in the tissues and covered with lymph. Coppez and Gunsberg maintain that the prognosis is more favorable with those bodies which are situated in the vitreous than with those entangled in the ciliary body or choroid. Goulden's results have been referred to (see page 296). If judicious efforts have failed to extract a foreign body from the interior of the eye, or if infection has proceeded beyond the reasonable hope of recovery, enucleation or evisceration usually is necessary.

If a particle of iron remains for some time in the eye, there is a deposit of iron pigment in its tissues which gives rise to a condition known as *siderosis bulbi*, characterized by a peculiar greenish-yellow or yellowish-brown discoloration of the iris and cornea, and a circle of brown dots beneath the capsule of the lens. The pigmentation may be due to the iron derived from the foreign body (xenogenous pigmentation) or to hemosiderin derived from blood (hematogenous pigmentation). Sometimes the iris regains its original color after removal of the foreign body and, occasionally, even if it remains within the eye.

Congenital pigmentation of the sclera (*melanosis sclerœ*) occurs both in spots and as a more diffuse discoloration. The spots are more common in the upper portion, and may be associated with pigment

changes in the iris and choroid. Pigment spots in the sclera have been observed in certain diseases—*e. g.*, Addison's disease—and sometimes are exactly symmetric, situated near the margin of the cornea.

Blue sclerotics may be associated with inherited syphilis and exhibit a remarkable hereditary transmission. In addition to the leaden color of the sclera, there may be conical cornea and congenital opacity of the cornea, that is, *embryotoxon*. Harman traced in five generations of one family 55 members, of whom 31 showed this congenital peculiarity. Transmission occurs through affected mothers. Persons with blue sclerotics are peculiarly liable to bony fractures.

CHAPTER IX

DISEASES OF THE IRIS

Congenital Anomalies.—*Heterochromia*, or the condition in which the color of one iris is different from that of the other, is a peculiarity which may be without pathologic significance, but in many instances the signs of cyclitis in the lighter colored eye are evident, and this eye is liable to cataract formation. It has been referred to on page 53.

Corectopia, a term applied to an eccentric position of the pupil, is not to be confounded with cases of true coloboma of the iris, presently to be described. The grade of corectopia may vary from a slight increase of the normal eccentric position of the pupil below and to the inner side, to those cases in which the whole pupil is displaced toward the border of the cornea. The latter variety is a very unusual phenomenon. This complete shifting of the normal position of the pupil has been ascribed either to an essential malformation or to the result of a fetal iritis. Both eyes may be affected symmetrically, and several members of the same family may present the defect.

Fig. 133.—Polycoria.

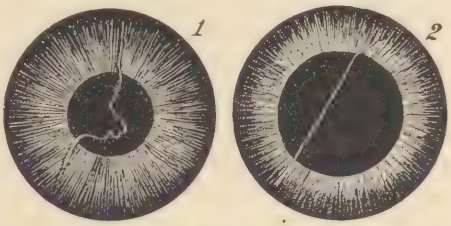


Fig. 134.—Persistent pupillary membrane: (1) Pupil contracted; (2) pupil dilated (Wickerkiewicz).

Polycoria, or a multiplicity of pupils, is a rare anomaly. The abnormal pupil or pupils may be situated in the immediate neighborhood of the normal pupil, separated from one another by a narrow band of iris tissue, or the increased number of pupils may be the result of crossing strands of persisting pupillary membrane (Fig. 133). An opening which exists at the ciliary margin of the iris has been described, and is probably due to a *congenital iridodialysis*.

Persistent pupillary membrane results from an incomplete resolution of the membrane which covers the anterior surface of the lens during fetal life, and which usually disappears in the seventh month, although it may remain as late as the end of intra-uterine life, and even in the first month after birth.

Accurately speaking, the pupillary membrane is a specialized portion of the *capsulopupillary* covering. The name of pupillary mem-

brane alone is applicable to those cases in which threads attached to the small circle of the iris pass diametrically or cord-wise across the pupil, to be inserted elsewhere in the *corona* (Fig. 134). Usually the fibers proceed from the anterior surface of the iris across the pupil, either singly or in groups of three or more strands. Sometimes the fibers remain separated; sometimes they grow together in front of the anterior capsule or unite in the form of a variously colored plaque, adherent to the capsule of the lens (*capsulopupillary membrane*). Persistent pupillary membrane is more common in one than in both eyes; of 68 cases observed by Stephenson, 13 were bilateral and 55 unilateral.

Capsulopupillary tags are not infrequently mistaken for the synechiæ due to iritis; indeed, the association of the two has been observed. No difficulty, however, should arise, because the normal action of the pupil is not impeded by the presence of these vestigial anomalies. The appearance is not often detected until some other disorder calls for an ophthalmoscopic examination, because vision is not seriously or at all im-

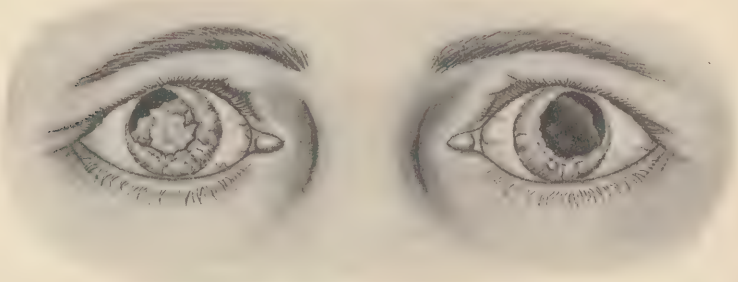


Fig. 135.—Bilateral coloboma of iris, upward and outward, and cataract (University Hospital).

paired. Oblique illumination or examination with a loupe or corneal microscope will readily demonstrate the remains of pupillary membrane.

Coloboma of the iris is a fissure of this membrane which in a general way resembles an artificial pupil made by iridectomy. The anomaly is more frequent in both eyes than in a single eye. Where the defect is unilateral, the anomaly is usually found on the left side. The situation of the fissure is generally downward or downward and inward. Exceptions to this rule have been observed; indeed, numerous atypical forms have been recorded, the defect being placed outward, inward, upward, down-and-out, up-and-in, and up-and-out.

The coloboma may extend across the whole iris (*complete coloboma*), or stop at a certain distance from the ciliary margin (*incomplete coloboma*). In addition, the so-called *pseudocoloboma* is described, which may be looked upon as a form of heterochromia of the iris, or indicates the last remains of the ocular fissure which is tending toward closure, and which appears as a small stripe, somewhat granular, and differentiated from the rest of the iris by its brighter color. In "bridge colo-

boma" the borders of the cleft are united by a narrow pigmented or colorless band of fibers.

Coloboma of the iris, often hereditary, is frequently associated with similar defects in the choroid, and also with microphthalmos, congenital cataract, fissure of the eyelids, lips, and palate. It has been attributed to an arrest of development, the result of incomplete closure of the *choroidal fissure;* but Lang and Treacher Collins believe that the defect is caused by a partial abnormal adhesion or late separation of the lens and cornea, the iris failing to develop in that portion of the area which is involved. If the abnormal adhesion or late separation is complete, irideremia results.

Irideremia, or congenital absence of the iris, occurs both in a *partial* and a *complete* form.

Total congenital irideremia is almost invariably bilateral. It is frequently associated with other anomalies of the globe—partial or complete cataract, dislocation of the lens, nystagmus, strabismus, departures from the normal curvature of the cornea, or annular opacities in its periphery and atrophy of the optic nerve. In a majority of instances there is a marked hereditary tendency.

Congenital ectropion of the uvea consists in a round mass of dark color projecting from the margin of the pupil, bending around to the anterior border of the iris. A similar formation is proper to the eye of the horse and is frequently seen in the cow. This appearance has sometimes been described as a *papilloma of the iris;* it is not, however, a neoplasm, but a congenital ectropion of the uvea.

Cysts, nevi, and *atrophies of the iris* occur as congenital defects, and *congenital aplasia of the anterior layers of the iris* has been observed.

Hyperemia of the iris is associated with several acute affections of the eye, for example, acute trachoma, purulent conjunctivitis, keratitis, scleritis, inflammations of the uveal tract, and traumas, and is a precursor of inflammation. Hence it is a symptom and not a disease of the iris.

Hyperemia of the iris is recognized by change in color, a blue iris becoming greenish; a brown iris, a reddish brown; by contraction of the pupil, which dilates sluggishly or not at all, to the changes of shade and light, and is slowly affected by a mydriatic, the effects of which are much less permanent than in the healthy iris; and by slight pericorneal injection.

The *treatment* consists in the management of the disease which has caused the hyperemia, and especially in the instillation of atropin.

Iritis.—Under the general term *iritis* are included various types of inflammation of the iris.

Causes.—Iritis may depend upon constitutional disorders, infections, toxins, and traumatism, or upon disease in other portions of the eye. Hence, it is usually stated that iritis is either *primary* or *secondary.* In point of fact, however, it is doubtful if the term "primary iritis" should be retained, inasmuch as iritis is probably never primary, but always secondary, in that it is one of the mainfestations of the action

of a toxin or an infection. To those cases of iritis which apparently originate independently of injury, or of an ocular or constitutional disorder, the name *idiopathic* was formerly applied, a term which should be eliminated, although, unfortunately, we are unable always to decide what exactly is the causative factor in each case. Iritis is also divided, according to its supposed etiology, into *syphilitic, rheumatic, gouty, gonorrheal, diabetic, tuberculous, scrofulous, septic, autotoxemic* or *toxemic, cachectic, traumatic,* and *sympathetic* iritis.

Symptoms.—1. *Change in the color* of the iris, in addition to loss of its natural luster and obscuration of the characteristic striated appearance.

2. *Pericorneal injection,* due to congestion of the non-perforating branches of the ciliary vessels (System II), producing the fine pink zone surrounding the cornea known as "ciliary congestion," or the "circumcorneal zone." In severe cases there may be distention of the posterior conjunctival vessels, and slight chemosis of the conjunctiva (see also page 49).

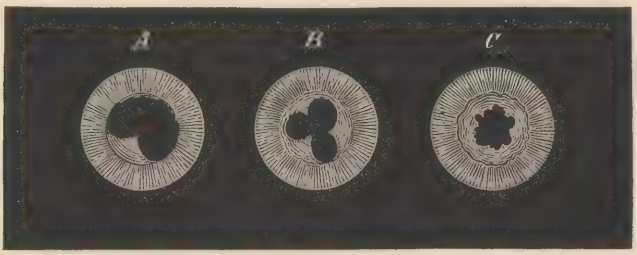


Fig. 136.—Various forms of posterior synechiæ: *A*, Single attachment; *B*, multiple attachment forming the so-called "ace-of-clubs" pupil; *C*, irregular annular attachments (Sichel).

3. *Miosis,* or contraction of the pupil, due partly to hyperemia and spasm of the sphincter, and partly to irritation of the peripheral nerve filaments. The reaction of the pupil to the influence of light and mydriatics is diminished or lost. In a certain number of cases, according to Herbert, there is slight dilatation of the pupil as compared with the unaffected eye. This primary dilatation of the pupil, with preservation of its reaction, is a noteworthy symptom in so-called rheumatic iritis (Krückmann; see also page 310).

4. The *formation of posterior synechiæ,* or attachments between the layer of pigment covering the posterior surface of the iris and the capsule of the lens. They are demonstrable by the instillation of a mydriatic, which will produce an irregular dilatation of the pupil, certain portions of the pupillary margin of the iris being held back by somewhat tongue-shaped projections attached to the lens-capsule, and may be readily studied by means of oblique illumination or with a loupe. The tags protruding into the pupil space usually have a brownish color.

The attachments may vary in size, firmness, and number; being either narrow and thread-like, broad and dense, single or multiple, or

even extending all around and pinning down the margin of the iris in an annular manner (annular posterior synechiæ). In association with the synechiæ there may be an exudation of false membrane covering the whole pupillary space (*pupillary membrane*).

5. *Irregularities in the surface of the iris*, due to local swellings, accumulations of exudation, deposits of fibrin, or the formation of nodules.

6. *Haziness of the cornea* or deposits upon its posterior surface. According to Friedenwald, the cornea is affected in every case of iritis, that is, there are deposits on Descemet's membrane or infiltrations in the substantia propria. The former are constant, the latter occasional.

7. *Changes in the character of the aqueous humor*—(1) Slight or considerable turbidity; (2) pus; (3) blood; and (4) occasionally exudation.

In addition to the symptoms just detailed there are *subjective* signs more or less peculiar to iritis.

1. *Pain.*—This is situated first in the eyeball, and is known as "ciliary pain," and second, in the brow and temple, sometimes quite sharply defined in the distribution of the supra-orbital nerve, very severe, throbbing, and stabbing in character, and with marked increase in severity during the night. Occasionally the nasal and infra-orbital regions are the painful areas. Pain in the teeth is not uncommon.

2. *Disturbance of Vision.*—This is in direct proportion to the amount of cloudiness which has occurred in the media. Decided impairment of visual acuteness denotes extension of the disease to the ciliary body or deeper structures.

During iritis, *transient myopia* and *astigmatism* are commonly present. Especially in the plastic types of the disease, even after full pupillary dilatation, an increase in the refractive power is demonstrable. Although there are changes in the corneal curvature, according to the late C. A. Oliver the bulk of the ametropic change in such cases is due to perversion of the lens action from *spastic accommodation* (Koller) as the result of ciliary irritation.

3. *Tenderness of the Globe.*—This is often present in uncomplicated iritis, but if severe, it suggests inflammation of the ciliary body.

4. *Photophobia and Lacrimation.*—These symptoms vary considerably in degree, being almost or quite absent in some varieties, and severe in those of acute and violent onset.

5. *Malaise*, fever, nausea, and marked depression occasionally are experienced by the patient, the last often being the result of prolonged pain and insomnia.

Diagnosis.—The salient symptoms of iritis just detailed are sufficient for the purpose of diagnosis; nevertheless, it is not uncommon to find a case of iritis mistaken for some other external inflammation, and the table on page 304 may be found useful.

A diffuse scleritis somewhat resembles in its color the zone of pericorneal injection more or less characteristic of iritis, which, indeed, may be a complicating symptom of this disease. Acute glaucoma bears some resemblance to acute iritis (for the distinguishing points see page 385.

Iritis.	Simple Conjunctivitis.	Phlyctenular Conjunctivitis.
1. Severe brow pain, worse at night.	Feeling of foreign body in the eye.	Acute general irritation.
2. Dim vision.	Vision usually unimpaired, unless secretion is very abundant.	Vision impaired by corneal involvement.
3. Fine pericorneal injection.	Coarse conjunctival injection.	Diffuse injection, with special lines of vessels running to phlyctenules.
4. Absence of secretion; some abnormal lacrimation.	Mucopurulent discharge; flakes of lymph.	Free lacrimation.
5. Sluggish or immobile pupil.	Pupil unaffected.	Pupil unaffected.
6. Iris discolored.	Iris unchanged in color.	Iris unchanged in color.
7. Abnormal reaction to mydriatic.	Normal reaction to mydriatic.	Normal reaction to mydriatic.
8. Severe photophobia exceptional.	Severe photophobia absent in simple cases.	Severe photophobia and blepharospasm.
9. Conjunctiva usually translucent; occasionally chemotic.	Conjunctiva opaque, velvety, and at times chemotic.	Conjunctiva translucent, bathed in tears.
10. Tenderness on pressure.	Tenderness not marked.	Tenderness not marked.
11. Posterior synechiæ.	No synechiæ.	No synechiæ.

Course, Complications, and Prognosis.—An iritis may pursue an acute course, reaching its termination in four to eight weeks, or be chronic from its onset and last, in a slow and insidious inflammation, for long periods of time. The termination of an iritis may be entirely favorable. The inflammatory adhesions disappear, and the iris regains complete mobility, only a few traces of iris pigment being seen on the capsule of the lens. On the other hand, more or less complete attachment causing distortion and inequality of the pupil (consult Fig. 136) may remain; or deposits of exudation may directly occlude the pupil and lie upon the capsule of the lens; or the tissue of the iris may show areas of atrophy and exhibit a bleached or grayish aspect.

The binding down of the iris throughout the whole extent of its pupillary edge, although the pupil itself remains clear, is denominated *exclusion* or *seclusion of the pupil;* if the pupil is filled in with opaque inflammatory deposit, the term *occlusion of the pupil* is applied. With extensive or annular synechiæ the angle of the anterior chamber becomes obliterated, the iris, owing to the exudation behind it, is bulged forward except around its pupillary margin, which is bound down, so that a crater-like depression is evident, and the appearance denominated *iris bombe* is developed. This leads to ncreased tension, secondary glaucoma, and even shrinking of the vitreous, detachment of the retina, and atrophy of the eyeball unless the communication between the anterior and posterior chambers of the eye is restored by operative measures (Figs. 137 and 138). Repeated attacks of iritis, as well as chronic inflammation of the iris, may cause *atrophy of the tissue.* In these circumstances the color of the iris is gray, the markings of its surface disappear, dilated blood-vessels and reddish patches appear, small holes develop in its tissue, the pupil margin is thin, and its tissue is friable

(Fig. 139). A form of progressive *atrophy of all layers of the iris* has been described (C. A. Wood, Harms, Zentmayer, and the author). Periods of increased intra-ocular tension are evident and the eyes become glaucomatous. Wood did not consider that glaucoma was an essential part of the affection, although it was the end process in his patient. The anatomic examination revealed, in addition to glaucoma and iris atrophy, a fibrinoplastic cyclitis.

Fig. 137.—Exclusion and occlusion of pupil with exudation behind iris, following gummatous iritis; compare with Fig. 138 (from a patient in the Philadelphia General Hospital).

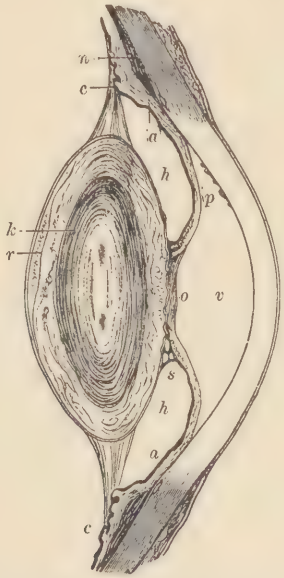


Fig. 138.—Exclusion and occlusion of the pupil. The iris is adherent by its entire pupillary margin to the lens. The posterior chamber (*h*) is thus made deeper, the anterior chamber (*v*) shallower, especially where the root of the iris (*a*) is pressed against the cornea. The retinal pigment is beginning to separate at *s*. The pupil is closed by an exudate membrane, *o*. In the lower part of the anterior chamber there is matter (*p*) precipitated upon the posterior surface of the cornea. The cortex of the lens has become cataractous (*r*); the nucleus (*k*) is unaltered (Fuchs).

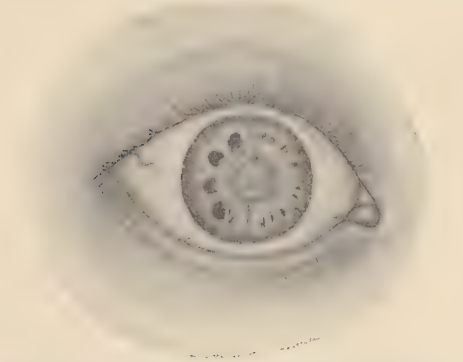


Fig. 139.—Atrophy of iris (from a patient in the University Hospital).

The following tissues of the eyes may become involved during the course of an iritis: The cornea (*keratitis punctata*); the ciliary body (*iridocyclitis*); the crystalline lens (*cataract*, especially *cataracta accreta*, in which the iris and lens are fastened together); the choroid (*iridochoroiditis*); the vitreous (*exudation into the vitreous*, *hyalitis*); and the optic nerve and retina (*hyperemia* or *neuroretinitis*). With these facts in mind, and with the tendency of certain types of the disease to relapse, a prognosis must be guarded, but in uncomplicated iritis, seen early

and properly treated, a perfect result may be obtained in the large majority of cases. *Relapses of iritis* are often attributed to posterior synechiæ, but, as Fuchs points out, are not due to them, but to the continuance of the cause of the affection.

Pathology.—Systematic writers at one time were accustomed to divide iritis into three varieties: *plastic*, *parenchymatous*, and *serous* iritis. A more accurate classification from the pathologic standpoint is *acute*, *chronic*, *purulent*, and *nodular* iritis. In general terms, in iritis it may be said that the iris is thickened and infiltrated with round cells, which are collected either along the line of the vessels or in circumscribed nodules. The vessel walls are thickened and small hemorrhages occur in the tissue. The exudation in the anterior chamber consists of round cells, mixed with fibrin and pigment granules. In many cases the inflammatory products are completely absorbed, but should they become abundant, they organize, forming a layer of connective tissue which covers the iris and binds it to the lens, occluding the pupil in the manner already described. The exudation in the iris likewise organizes, and the atrophic iris shows obliterated and thickened vessels, clumping of pigment granules, and an entire absence of iris-stroma. *Purulent* iritis due to infection with micro-organisms is followed by panophthalmitis. This infection may come from without, as it occurs, for example, in perforating injuries, and is called *exogenous infection;* or from within, as, for example, in metastatic processes, and is called *endogenous infection*. In *nodular* iritis the nodes are formed of aggregations of lymphocytes. It is often difficult to distinguish between the varieties of iritis from the histologic standpoint, as they merge one into the other. If the exudation is poor in cells and fibrin, and the iris tissue shows cellular infiltration, there is serous iritis (not to be confounded with the cyclitis clinically called serous iritis); if the exudation is rich in fibrin but poor in cells, and the iris tissue markedly infiltrated, there is *fibrinous iritis;* if with an exudation rich in cells and fibrin there is extensive infiltration of the iris tissue, with mononuclear and multinuclear leukocytes, there is *purulent iritis* (Ginsberg).

Treatment.—The description of the treatment is reserved for the subsequent sections devoted to the particular consideration of the various types of iritis which follow.

Iritis and Its Types.—In the acute form of the disease the salient symptoms of iritis already recorded (see page 302) are present. Not only may the ordinary attachments form between the iris and the capsule of the lens, but a plastic exudation may cover the pupil-space with a false membrane, and the adhesions between the iris and the lens-capsule may be unusually firm and unyielding. To this form of iritis the descriptive term *plastic* is sometimes applied. In some cases a gelatin-like mass is deposited in the anterior chamber, and its appearance has been compared to that of a dislocated lens in the same position (*fibrinous* or *spongy iritis*). In various forms iritis is seen in—

1. *Syphilis—Syphilitic Iritis.*—The percentage of patients with syphilis who acquire iritis during the course of the disease varies from

0.42 to 5.37, according to the different authorities, but among cases of iritis, syphilis has been found to be the cause in from 30 to 60 per cent. (Alexander). In other words, syphilis is usually regarded as the most common cause of iritis. It may appear between the second and the ninth month after the initial lesion, or may be delayed until the eighteenth month. Occasionally it arises at a very late period in syphilis —that is to say, during the period of so-called tertiary manifestations, either as a primary iritis, as a relapse, or in one of the forms presently to be described. The lesions are due to the influence of syphilitic virus, that is, to the lodgment and activities of the Spirochæta pallida, which has been found in the aqueous humor of an eye with acute syphilitic iritis (Zur Nedden, Stephenson).

The clinical manifestations of syphilitic iritis vary. They may be those of ordinary acute iritis without lesions, which of themselves do not justify the diagnosis of syphilis, and syphilis as the etiologic factor can be established only by the history of the case, by the

Fig. 140.—Papules in syphilitic iritis (from a patient in the Philadelphia General Hospital).

Fig. 141.—True syphilitic iritis with large nodes in iris; about seventh month of the disease.

therapeutic test, or by the serum reaction of Wassermann. According to Krückmann, an early manifestation of syphilis in the iris, which may appear in the sixth week after primary infection, is *roseola*, characterized by overfilling of superficially placed vessel loops, which arise and disappeared quickly. The color is bright red in contrast to the copper tint of the skin eruption. Roseola may in later stages be the forerunner of papules. Localized sphincter lesions always suggest the influence of syphilis (Krückmann), and with the development of papules a form of iritis appears which yields characteristic, if not pathognomonic, signs of its origin. In the inflamed iris there appear one or more yellowish, reddish-yellow, or reddish-brown nodules, varying in size from a hemp-seed to a small pea, situated at the pupillary or ciliary border, or occasionally between the two in the iris tissue, although Fuchs maintains that they do not arise in the midbreadth of the iris. They vary in number from one to four, the intervening tissue being comparatively unaffected, and belong, in spite of their resemblance to gummas, with which at one time they were confused, to a comparatively early period of syphilis—

that is, to the period indicated in the previous paragraph. Under the influence of treatment they are gradually absorbed without leaving very marked scars, although a certain amount of atrophy of the iris tissue on close examination will be found marking their former situation. This form of iritis is sometimes called *true syphilitic iritis* or *syphilitic parenchymatous iritis*. It is also known under the terms *iritis papulosa*, or *condylomatosa*.[1] These names have originated because the small nodules in the iris have been compared to papular syphilids and condylomata, inasmuch as they belong in the same stage of syphilis with these manifestations. They clearly differentiate themselves from gumma, not only by the date of their appearance, but because they do not caseate or break down and suppurate.

Even where these distinct nodules are not present in syphilitic iritis, careful examination of the iris will frequently show localized discolored swellings in the edematous iris tissue, and usually broad and thick synechiæ, formed by a union of the iris tissue with lens-capsule, and not merely an adhesion of the posterior epithelium, and, as Fuchs has shown, microscopic investigation indicates that these nodules are always present, but are sometimes so small that they have not sufficiently elevated the iris tissue to reveal their presence to ordinary clinical methods of examination. To this form of iritis the name *syphilitic* or *luetic fibrinous iritis* is sometimes given.

The disposition and character of the papules which develop on the iris as the result of syphilis have been particularly studied by Krückmann, who among the early varieties describes superficial and deep-seated *small iris-syphilids*, the chief situation of the latter being in the vessels system of the sphincter and its immediate neighborhood, and *medium-sized papules* which develop under the anterior stroma layer. The papules of an early stage of syphilis often have a reddish color, while those of later periods of the disease are yellow or grayish yellow and more decidedly circumscribed, owing to the absence of edema. A rare manifestation is the eruption of the papules in a group formation.

Gummatous iritis or, more properly, *gumma of the iris* occurs, appearing, according to Alexander, almost constantly at the ciliary border. The lesion is solitary, of the size of a pea or small nut, and grows toward the ciliary body, disappearing through fatty degeneration, leaving behind a permanent scar, or *atrophy of the iris*. Such a manifestation, strictly localized in the iris, is extremely rare. It appears, if at all, in the so-called tertiary period of syphilis, or that period in which gummas in other organs are found.

In syphilitic iritis both eyes are attacked, one a little later than its fellow; occasionally the onset is simultaneous. The course usually is acute, and after thorough cure relapses are not common. Sometimes the disease assumes a subacute type, or may be so prolonged in its course and complications as to justify the term *chronic iritis*.

[1] Krückmann objects to the term "iritis condylomatosa" because there is no accurate comparison to be made between condyloma of the skin and the papules of the iris in syphilis.

Acute iritis of the so-called plastic type is rare in newborn infants of syphilitic heritage, but has been described in children with *inherited syphilis*, from the second to the fifteenth month. Acute iritis in children in the first months of life, and also in later childhood years, usually is the result of hereditary syphilis. A late manifestation appears in the form of an iridocyclitis of the so-called serous type, the involvement of the entire uveal tract being evident by the manifestations of the signs of uveitis, which are elsewhere described.

Treatment.—The most important local drug in this as in other forms of iritis is atropin sulphate, gr. iv to f℥j (0.26 gm. to 30 c.c.), several drops of the solution to be instilled in the conjunctival culdesac every three or four hours. Mydriasis should be maintained until all ciliary irritation has subsided and during the period of changes in the refractive power of the eye (see page 303).

Pain is relieved and at the same time congestion is diminished, thus rendering the mydriatic action of the atropin more certain, by leeching the temple—one to three Swedish leeches being applied near the line of the hair, or blood is drawn by an artificial leech. In the absence of a regular Heurteloup, this may be accomplished by making an incision in the temple with a scalpel and using a small cupping-glass, to which a piston is attached for exhausting the air. Should atropin not be tolerated, hyoscyamin, scopolamin, or duboisin may be substituted (see page 117).

The constant use of atropin leads to disagreeable dryness of the throat. This may be obviated in part by compressing the tear-duct after each application. It may be relieved by giving the patient a gargle made of equal parts of iced water and a strong decoction of coffee.

Pain is further relieved by the application of moist or dry heat; the latter is best made by means of cotton batting which is held before a fire and then laid upon the affected eye, to be replaced by a freshly heated mass as soon as cooling occurs, or with a Japanese stove or hot box. Moist hot applications are more efficient if a pad of surgical gauze is steeped in the following solution: Acetate of lead, ʒj (3.9 gm.); powdered opium, ʒss (15.5 gm.); boiling water, Oj (473.11 c.c.) (Randolph). Dionin in 5 to 10 per cent. solution is valuable on account of its lymphagogue and analgesic action, which is increased by the addition of a 2 per cent. solution of holocain. With the use of *high-frequency* currents for the relief of the pain of iritis the author has had no experience.

The best constitutional treatment is some form of mercury, either the protiodid, blue mass, or calomel, given, as in syphilis generally, just short of the point of salivation, and continued for many weeks even after all acute symptoms have subsided. Inunctions of unguentum hydrargyrum are advantageously employed, preceded by a hot bath or diaphoresis with the aid of a hot chamber. If inunctions are properly given they represent a most satisfactory method of administering mercury, and usually ʒj (3.9 gm.) of the ointment may be daily rubbed into the skin. Hypodermic or, rather, intramuscular injections of mercury, particularly mercuric chlorid or salicylate of mercury (of

mercuric chlorid the dose may be from $\frac{1}{16}$ to $\frac{1}{8}$ grain—0.00405–0.008 gm.; of salicylate of mercury, $\frac{1}{2}$ grain—0.0324 gm.), are strongly advocated by some surgeons.

In syphilitic iritis *salvarsan* produces the most favorable results, and under its influence the lesions disappear with astonishing rapidity. If the Wassermann test is positive, other things being equal, a dose of 0.6 gram should be given intravenously (in women 0.4 gram is usually sufficient). At the expiration of three weeks, the Wassermann test being still positive, this dose may be repeated. During the intervals mercury or iodid of potassium should be administered.

Clinical trial of *neosalvarsan* indicates that it equals *salvarsan* in efficiency and that it is less toxic. In arsenic content 0.6 gram of salvarsan is equivalent to 0.9 gram of neosalvarsan. The injections may be given at comparatively frequent intervals, the dose being regulated according to circumstances and effects, as often as once in ten days or two weeks.

In old syphilitics with much cachexia, in whom a plastic iritis improperly treated in the early period has relapsed, it is not always wise or possible to induce active mercurialization. For them bichlorid combined with the tincture of iron is a suitable remedy. Subconjunctival injections of bichlorid of mercury (2 to 4 drops of a 1 : 2000 solution) are efficient, but painful. Acoin added to the injection relieves the pain. In place of the bichlorid solution one of cyanid of mercury (1 : 5000) is now advised by Darier. Equally good results are obtained with 5- to 15-minim (0.3–0.92 c.c.) injections of physiologic salt solution if there is not too much circulatory stasis. The injections may be given every second or third day, and should be followed by light massage of the eyeball. Usually they are not necessary if active constitutional treatment (salvarsan, mercury) has been carefully carried out.

2. *Rheumatism—Rheumatic Iritis.*—According to Krückmann, rheumatic iritis is apt to begin with conjunctival hyperemia or a non-bacterial conjunctivitis; at first, in some cases, there may be mydriasis with preservation of the pupil reflexes and congestion of some of the radially placed larger iris vessels, followed by a sudden increase of the iris injection and the appearance of pericorneal injection, pupil immobility, fibrous exudation into the superficial stroma layer, and fine deposits on the posterior surface of the cornea. The vitreous remains clear.

The association of iritis with *acute rheumatism* (acute rheumatic fever), however, must be exceedingly uncommon, if it ever occurs. Indeed, as Krückmann maintains, the so-called rheumatic iritis must be sharply separated from those iritic involvements which follow or accompany acute joint rheumatism, and which depend upon metastasis of staphylococci or streptococci proceeding from purulent processes in the joints. Paine and Poyton have isolated a diplococcus which they regard as the specific cause of rheumatic fever, and with which experimentally they were able to produce an iridocyclitis which was regarded as a true rheumatic iridocyclitis. In so far as acute rheuma-

tism is concerned these observations have been confirmed by Rosenow, whose investigations also indicate that muscular rheumatism (see below) may be due to a closely related streptococcus.

Because the relation of rheumatism to the development of iritis is a vague one, and because the general "rheumatic" conditions—myalgia, joint lesions, etc.—may be toxic in origin, T. Harrison Butler suggests that the ocular manifestation should be denominated *autotoxemic* or *toxemic iritis*, and that the term "rheumatic" should be abandoned.

3. *Autotoxemic Iritis* (*Iritis with Disorders of Nutrition or Constitutional Disorders*).—Notably between the ages of twenty and fifty, but also at later periods of life, either coincident or not with affections which are usually classified as chronic rheumatism, chronic joint rheumatism, or muscular rheumatism—*i. e.*, myalgia manifesting itself as lumbago, pleurodynia, or pain in various groups of muscles (usually classified by systematic writers as "constitutional diseases" or "diseases of nutrition")—iritis is not uncommon. In Butler's statistics this type of iritis or iridocyclitis constitutes 6 per cent. It varies considerably in the aggressiveness of its symptoms. Not uncommonly these are severe, with much pericorneal injection, acute pain, greater usually than in syphilitic cases, and tenderness of the globe. Frequently only one eye is affected; the inflammation rarely is simultaneously symmetric. The second iris may be affected in like manner after a longer or shorter interval. From the subjects of this form of iritis, if none of the various types of polyarthritis or myalgia is present, a history of such an affection can usually be obtained; sometimes the history develops the fact that the patient has suffered from sciatica, or crural, musculospinal, and other forms of neuritis.

Relapses are frequent, in this particular differing from syphilitic plastic iritis, and a patient once having had an attack of this type of iritis (often in its manifestations an iridocyclitis) is liable at intervals of months or even years again to be attacked. If treatment is begun early, even in recurring attacks, perfect cure may be expected.

The frequent relapses of some varieties of the affection have given rise to the term *recurrent iritis*.

A form of iritis exists, aptly called *quiet iritis* (Hutchinson), in which there is no pain or ciliary congestion; it is practically non-adhesive, the only subjective symptom being the progressive dimness of vision, which leads to its discovery, and which is caused by so-called chronic rheumatism or inherited arthritic tendency in a majority of cases, but which may also depend upon syphilis. A variety of quiet iritis in which the lesions are said to be confined to the posterior layer of the iris has been described by Grandclément under the name "Uvéite irienne." It occurs usually in women during the period of uterine activity; frequently its subjects are anemic. Grandclément failed to associate it with any local or general malady.

A severe and sometimes destructive form of iritis may accompany *arthritis deformans*. There is a variety of iritis characterized by warty-looking translucent excrescences at the pupil margin, generally occur-

ring in women, to which Doyne has given the name *guttate iritis*. (Compare this section with pages 324–326.)

With two constitutional diseases or disorders of nutrition iritis or iridocyclitis is not infrequently associated, namely, gout and diabetes.

Gouty iritis, as it is usually called, occurs in the subjects of gout, irregular gout (often referred to with questionable accuracy as gouty, lithemic, or uric acid diathesis), and in the members of gouty families. It probably depends upon the defective nitrogen metabolism which underlies gout, although this origin is not accepted by some authors. It tends to relapse, to attack one eye at a time; the superficial layers of the iris are especially affected. It may precede a gouty attack elsewhere in the body. A form of iritis, insidious in character and destructive in tendency, almost invariably associated with disease of the vitreous, occasionally occurs in children of gouty parents. These children, according to the late Mr. Hutchinson, have a peculiar squareness of build, heavy features, florid complexions, and feebleness of circulation in the extremities. Less frequently iritis (sometimes purulent), sometimes associated with fibrous deposits in the pupil space and with hemorrhage in the anterior chamber, develops in the subjects of diabetes; it is ordinarily described as *diabetic iritis*. Its frequency among diabetics has varied from 1.5 to 5 per cent.

Treatment.—The use of atropin in the manner already described is of paramount importance. For it scopolamin, gr. ij to f℥j (0.13 gm. to 30 c.c.), may be substituted, or the two drugs may be combined. Leeches and moist and dry heat will help to relieve the pain, and, at the proper stage, subconjunctival injections of salt solution. Much comfort often results from the administration at night of $\frac{1}{100}$ grain (0.00065 gm.) of hyoscin, but morphin or codein, if given at all, must be administered with great caution lest the patient form a drug habit. Rubbing the brow with an ointment of mercury and belladonna is of some service. Dionin (5 per cent.) and holocain (2 per cent.) act well in relieving pain.

Much reliance may be placed upon salicylic acid, salicylate of sodium, salicylate of strontium, and aspirin; of these remedies, salicylate of sodium is the best; indeed, it relieves the pain of any form of iritis. It should be exhibited in full doses, 60 to 80 grains (3.9–5.2 gm.) during the first twenty-four hours, and afterward the amount gradually lessened.

The tendency to recurrence requires preventive treatment in the form of regulated diet, the use of mineral waters, and proper attention to change of clothing, according to the vicissitudes of the climate. A course of treatment at some establishment connected with the various medicinal springs is of great benefit. In all forms of iritis, especially in the autotoxemic types, the nasopharynx, the tonsils, the accessory sinuses, and the buccal mucous membrane should be carefully examined for focal infection, which may be the cause of the toxemia of which the patient is the subject as well as of the iritis (see also page 324). Intestinal putrefaction may be present and must be corrected.

If the iritis assumes a chronic type, or if there has been exudation of lymph or involvement of the ciliary body, mercury and iodid of potassium may be exhibited. After the inflammatory signs of iritis have thoroughly subsided and the eye is quiet, the refractive error should be thoroughly corrected and the glasses worn constantly, because there is no doubt that this plan of treatment distinctly checks the tendency to relapse.

Should gout be determined to be the underlying cause, the usual treatment of this affection—dietetic and medicinal—is indicated, in addition to the local measures. Medicinal springs treatment is especially valuable. The subjects of gout must be treated on general principles.

4. *Gonorrhea—Gonorrheal Iritis.*—This is a form of iritis (often a severe iridocyclitis), chiefly plastic in character, does not necessarily coincide with nor immediately follow the gonorrheal attack; an arthritis of the knee, or sometimes of the ankle, intervenes; sometimes arthritis and iritis occur at the same time, and sometimes the iritis precedes the arthritis. Brailey has seen it assume a gelatinous type. This disease is much more common than has usually been supposed, and there may be a long interval between the acquisition of the gonorrhea and of the iritis—frequently several years and even as long as thirty years. The blood should be examined for a complement-fixation reaction, and the posterior urethra for gonococci. It should be remembered that an ordinary iritis in a person with syphilis may be due to a gonorrhea from which he has also suffered. In many instances so-called rheumatic iritis is really gonorrheal in origin; indeed, William Lang believes that the gonococcus is the most frequent cause of plastic iritis. The affection is due to the influence of the gonococci and their toxins on the iris. The presence of these micro-organisms in the anterior chamber has been demonstrated (Sidler-Huguenin). The disease is attended with severe pain, in addition to the usual symptoms of iritis, and its chief manifestations are in the superficial layers of the iris. It may relapse with each new attack of gonorrhea.

Treatment.—The local use of atropin, etc., is indicated. If the urethra is inflamed, this must receive attention. Iodid of potassium may be tried, and mercury, if there is much exudation. Relief will follow profuse sweats by means of pilocarpin given hypodermically or with the aid of an ordinary hot chamber or cabinet; indeed, these remedies are of great value in other varieties of stubborn iritis. Subconjunctival injections of salt and of cyanid of mercury have been advocated, and dionin in the usual manner may be employed. Excellent results, in the author's experience, follow the administration of gonococcic vaccine (Neisser bacterin). Large doses are tolerated: 50,000,000 to 100,000,000 organisms may be injected at intervals of three to seven days. Even larger doses are advocated by William Lang (200,000,000 to 500,000,000 at intervals of a week). John Weeks, however, thinks smaller doses (2,500,000 to 50,000,000) produce equally good results.

Iritis Secondary to Mucous Membrane Infection.—Iritis (more often iridocyclitis or uveitis) caused by infection of mucous membranes is of frequent occurrence. The primary source of such infection most frequently is a chronic septic process in the mouth (pyorrhœa alveolaris), in the teeth (tooth-root abscesses), in the tonsil, in the nasopharynx, in the accessory nasal sinuses, in the stomach and intestines, in the urethra (see page 313), in the uterine cavity, the prostate, and in the skin (boils, furuncles, etc.).

Treatment.—In all cases of iritis and iridocyclitis the regions named must be carefully searched for disease and persistently treated if it is present. The usual local ocular treatment is indicated. Vaccine therapy (usually the staphylococcus is the active organism, but others may be potent) may be efficient. (For additional information, see pages 327, 328.)

In malaria a *periodic iritis* with hypopyon has been described, and somewhat analogous to this is another periodic iritis, or iridocyclitis, which has been seen before each menstrual period (*iritis catamenalis*), perhaps due to abnormalities in the uterine discharge. Fuchs reports severe iridocyclitis in association with general alopecia. A relation between nephritis and iritis has been described. Sometimes plastic iritis of moderate grade is found in elderly persons for which no cause can be found; to these types of iritis formerly the name "idiopathic" was applied.

Scrofulous iritis occurs usually in children and young persons of scrofulous habit. In some respects it resembles inherited syphilitic iritis. Nodules of lardaceous appearance may also form. *Tuberculous iritis* is described on page 317.

Infectious disease iritis is seen in association with recurrent fever, variola, pneumonia, pertussis, parotitis, tonsillitis, herpes zoster, cerebrospinal meningitis, influenza, dysentery, typhus and typhoid fever, and a *purulent iritis*, as the result of embolism, occurs in the course of septicemia after puerperal fever, and in pyemia.

The management of such cases depends upon general principles, the free use of quinin and stimulants being appropriate in purulent iritis.

Traumatic iritis occurs as the result of an injury, either accidentally inflicted or due to an operation—*e. g.*, cataract extraction.[1] In this category are placed, also, those cases of iritis which follow discission of the lens and which depend upon swelling of the cortical material and infection conveyed through the wound. The iritis excited by foreign bodies—for instance, metallic particles—embedded in the iris has been ascribed to a chemical as well as to infective action.

Treatment.—The usual local measures are advisable. Iced compresses are advantageous, and the internal administration of mercury and the salicylates is indicated.

Sympathetic iritis (see page 333).

[1] Spongy iritis (see page 306) is occasionally seen after cataract extraction (Knapp). Plastic iritis has been ascribed to the action of strong solutions of eserin (*eserin iritis*); but it is doubtful if the drug could produce such an effect in a healthy eye.

Secondary iritis, independently of the fact that in a certain sense all types of iritis are secondary, may depend upon exogenous sepsis, for example, that which results from an infected corneal wound (see Traumatic Iritis) or upon exogenous toxemia, for instance, that which proceeds from an infected corneal ulcer (see page 306).

Scleritis of the deep variety is often associated with iritis. More rarely the primary disease begins deep in the eye—*e. g.*, in detachment of the retina. The presence of intra-ocular tumors, vitreous exudations, or retinal hemorrhages may occasion a secondary iritis.

Serous Iritis.—At one time it was the custom to describe a form of iritis characterized by a serous or, more commonly, a seroplastic exudation, deepening of the anterior chamber, slight dilatation of the pupil, haziness of the cornea and aqueous humor, and a precipitate of opaque dots upon the posterior surface of the cornea, generally arranged in a triangular manner, with the apex pointing upward, with the term *serous iritis*, or *serous iritis* and *keratitis punctata* (see page 323). Both of these terms are inappropriate, the one indicating purely a symptom of a disease, and the other an unproved pathologic condition. For a full consideration of this matter, see page 323.

Chronic Iritis.—Any type of iritis may assume an acute, subacute, or chronic course; if the last, no additional symptoms occur, but those ordinarily present are modified by the chronicity of the stages.

In addition to the chronic type of an ordinary iritis there remains to be described one which has received the name *plastic iridochoroiditis*, because of co-existing disease of the choroid and vitreous, leading to the formation of a secondary cataract. This disease occurs in adults, often without assignable cause, is symmetric, and proceeds steadily in a tendency destructive to the nutrition of the eye (see also page 326).

The *treatment* of the latter condition is unsatisfactory, alteratives, tonics, and operative measures often meeting with indifferent success.

Operative Treatment in Iritis.—Paracentesis of the cornea may be needed to reduce continued elevation of tension in some forms of iritis, and has been advised as a therapeutic measure in some varieties of iridocyclitis. Should inflammation of the iris and hypopyon exist, the treatment already described (see page 254) is required.

Iridectomy is often recommended for the relief of recurrent iritis, or in an iritis which refuses to heal completely, some ciliary injection and irritability remaining. Those cases which present the least change in the iris, in which the aqueous humor is clear and the tension is not subnormal, are most likely to yield a good result. Iridectomy in recurrent iritis does not insure the patient against future attacks, and represents a method of treatment which, in the author's experience, usually is most unsatisfactory.

In chronic iritis, however, circular posterior synechiæ and bulging of the iris are important indications for the operation. Determined rise of tension and threatening glaucoma, in any circumstances, furnish imperative reasons for its performance. According to the late Mr. Nettleship, keratitis punctata, chronic thickening of the iris with very

extensive attachments, the existence of myopia, a tendency to spontaneous bleeding, and hypopyon render the operation less desirable; if the tension is below the normal, the operation may be followed by bleeding and shrinking of the eyeball; occasionally, even in these circumstances, excellent results are achieved. If, however, a map of the visual field, sometimes obtainable only with a small point of light, indicates extensive deep disease, the chances of operative success are notably lessened and the operation may be contraindicated.

An iridectomy is performed to secure one or all of three ends: (1) Prevention of recurring attacks; (2) re-establishment of the communication between the anterior and posterior chambers of the eye, and thereby improvement in nutrition and aversion of threatened glaucoma; (3) improvement in vision by the substitution of an artificial pupil for one that has been occluded or excluded.

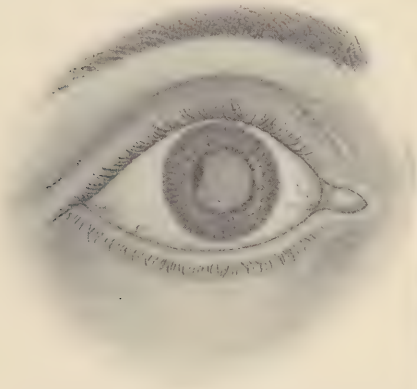


Fig. 142.—Cyst growing from posterior surface of the iris advancing into the anterior chamber (from a patient in the University Hospital).

Fig. 143.—Cyst of the iris following traumatism (from a patient in the University Hospital).

That portion of the iris should be selected for excision which is least changed and least bound down by adhesions.

Posterior synechiæ remaining after the acute symptoms of iritis have subsided have been regarded as a cause of relapse or recurrence, and, although this has not been proved (compare with page 306), several operations have been devised for severing such attachments, to which the general term *corelysis* has been applied.

Tumors of the Iris.—**Cysts.**—Cysts having transparent, delicate walls lined with pavement epithelium (*serous cysts; retention cysts*) may be congenital or may develop in the iris as the result of an injury. They are due to closure of the mouth of an iris crypt and its distention with the retained fluid. Cysts formed by a separation of the two layers of the pigmented retinal epithelium at the back of the iris (*cysts of the retinal epithelium*) are due, according to Treacher Collins, to interference with the lymph-current of the iris. *Multiple cysts* may develop

on the posterior surface of the iris (Pagenstecher, Fuchs, Wintersteiner). Such a cyst may have a brownish color and be mistaken for a malignant tumor. A *parasitic cyst*—that is, one due to *cysticercus* in the iris—has been reported. Implantation of a cilium, or of superficial epithelium, in the anterior chamber may be the starting-point of an *epithelial, pearl-like tumor* (*pearl-cysts* or *cholesteatoma*), essentially cystic, with a lining of laminated epithelium and semisolid contents of degenerated epithelial cells and fat-globules (F. R. Cross and E. T. Collins). Traumatic cysts, which owe their origin to the intrusion through a wound of corneo-conjunctival epithelium, which proliferates, are divided by J. Meller into *iris-cysts proper*, which are situated entirely within the iris tissue, *iris-chamber cysts*, which are situated partly in the iris and partly in the anterior chamber, and *wall-chamber cysts*, which are so situated that the iris forms only part of their boundary wall.

A cyst may be minute, or grow and fill the anterior chamber; both eyes may be affected, and some instances of multiple iris-cysts are on record (see page 316). A cyst may cause iridochoroiditis by pressure. An attempt should be made to remove it through an incision, the growth and surrounding iris being seized, drawn out, and excised.

Tubercle of the Iris (*Tuberculous Iritis*).—In a certain number of persons, usually between the ages of five and twenty-five (Pechin), small, grayish-red or yellowish nodules develop at the margin of the pupil or at its ciliary border, bearing great similarity in their external appearance to miliary growths (*disseminated miliary tubercle of the iris*). The nodules are usually 2 to 3 mm. in diameter, and may be situated close to the anterior surface of the iris or deep in its stroma. Two terminations have been observed: the growths may develop slowly and finally be absorbed and disappear, posterior synechiæ remaining at their points of origin (*attenuated tubercle of iris*, Leber); or successive developments of new nodules may lead to a plastic inflammation of the iris and ciliary body, and involvement of the cornea (*keratitis punctata*), and cause perforation at the corneoscleral junction, and shrinking of the eyeball. In these circumstances tubercle of the iris appears in the form of an iritis. In some cases of tuberculous iritis the nodules are ill-defined, being situated within the inflamed and thickened iris.

Tubercle of the iris also occurs in a *confluent* or *conglomerate* form, a yellowish nodule growing from the periphery of the iris, covered, it may be, with smaller bodies. The tendency of this growth is to increase, to perforate the eye, and to cause a general dissemination of tubercle.

The average age of persons affected with primary tuberculosis of the iris is twelve years; one or both eyes may be affected, more commonly the former. Although the patients may present no other signs of tuberculosis, this, and in a fatal form, may become a sequence. Sometimes the affection of the iris is secondary to the general disease. Bacilli and giant-cells may be found in these growths, proving their true nature, or the diagnosis must rest upon the results of inoculation of a rabbit's or guinea-pig's anterior chamber with a fragment of the suspected tissue, or upon tests with tuberculin, either by means of

subcutaneous injection or cutaneous vaccination. For diagnostic purposes Koch's old tuberculin may be used in gradually increasing doses, beginning with 1 mg. and with two-day intervals increasing to 5 mg.

Treatment.—Removal of a tubercle of the iris is almost always unsuccessful, except in some varieties of attenuated tuberculosis. Hence, if the disease is attacked from the operative standpoint, enucleation has been recommended. Before radical surgical procedures are adopted there should be a thorough trial of the therapeutic value of tuberculin. Von Hippel, using *tuberculin T. R.*, begins with the dose of $\frac{1}{5000}$ mg., and gradually increases to $\frac{1}{500}$ mg., and even to $\frac{1}{50}$ mg. In some cases the dose is further increased from $\frac{1}{5}$ mg. to $\frac{5}{5}$; *i. e.*, 1 mg. by $\frac{1}{5}$ mg. at each injection. A bouillon filtrate of tuberculin obtained from the Saranac Laboratory, with the initial dose of 0.0001 mg., is recommended by G. S. Derby. The author's results with tuberculin as a therapeutic agent have been most satisfactory. He has also used with satisfaction tuberculin "Old." The five dilutions contain respectively 1 : 1000 mg., 1 : 100 mg., 1 : 10 mg., 1 mg., and 10 mg. The dose of each dilution is 2 mg., and is progressively increased until 20 minims are injected. Following this, the next series is begun. The introduction of iodoform into the anterior chamber has been tried.

Sarcoma of the iris is rare as a primary growth. It has been well studied in this country by Veasey, and more recently by C. A. Wood and Brown Pusey. Iris sarcoma is more common in the latter half of life—that is, after thirty years—than at an earlier period, although a few cases have been reported in the first decade of life. Females are more often affected than males; the lower half of the iris is the primary seat of the growth in a large percentage of the cases. A few instances of bilateral iris sarcomas are on record. The first stage of the tumor's growth is slow, and may last for months and even years; in the second and later stages there is rapid increase in size, with pain, hemorrhage, etc., and, finally, rupture of the globe. Usually the tumor is pigmented; rarely a leukosarcoma of the iris develops. Histologically, small round and small spindle cells are the predominating forms. The growth must be differentiated from melanoma, tubercle, and gumma. A few iris sarcomas have been successfully removed by iridectomy (Thorington); but Wood and Pusey are emphatic in their advice that the globe shall be enucleated as soon as the diagnosis is certainly established.

Melanoma of the iris is a dark tumor, developed from the pigment stroma of the iris, and although commonly passive and innocuous, is occasionally the precursor of sarcoma. Melanomas also occur at the pupillary margin of the iris, where they develop from the retinal pigmented cells.

Rare forms of iris tumor are vascular growths (nevi), leprosy nodules, and myomas. It is more than doubtful if primary carcinoma of the iris occurs; it may develop as a secondary growth, as also may glioma.

Injuries to the Iris.—Wounds.—An incised wound limited to the iris does not necessarily produce serious results. It will be followed by blood in the anterior chamber, which in course of time is absorbed.

Wounds, however, are rarely limited to the iris, but having penetrated the eyeball through the cornea or ciliary region, may cause sympathetic irritation, or injure the lens and produce traumatic cataract.

In the first instance atropin, to secure physiologic rest of the iris, and a compressing bandage will lead to a speedy cure; in the other instances the extent and position of the wound will determine the necessity for enucleation or for the treatment applicable to traumatic iritis.

Foreign Bodies.—A foreign body may penetrate the cornea and lodge in the iris, or, having partially penetrated the cornea, may be pushed through it in the efforts at dislodgment and become entangled in the iris.

An opening is made with a broad needle or narrow keratome at the corneoscleral junction, eserin having been previously instilled, and a pair of forceps passed into the wound, with which the body is seized, or a small loop of platinum wire may be slipped beneath the fragment, by means of which it is withdrawn. If this is not possible, the piece of iris

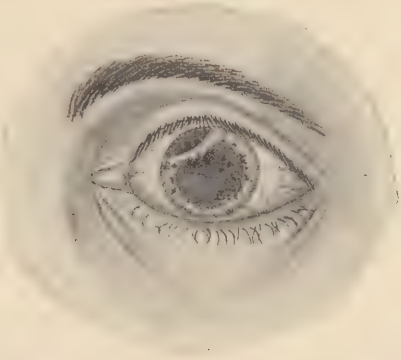


Fig. 144.—Iridodialysis and partial cataract; pupil dilated.

in which the substance is entangled may be drawn through the wound and excised. If the body is composed of steel or iron, it can be dislodged with a magnet.

Blows upon the eye may cause the following lesions:

Iridodialysis is a rupture of the ciliary attachment of the iris (ligamentum pectinatum). By this means an opening is produced comparable to a false pupil; it may be detected by the red reflex which shines through the artificial aperture, usually somewhat semilunar shaped, situated in the periphery of the iris at the corneoscleral margin (Fig. 144). This may be quite small or involve more than half the circumference. The injury may produce other lesions—for example, cataract.

In a few instances reattachment of the ruptured fibers has taken place under the favoring influence of atropin, which should be vigorously instilled. Ordinarily the lesion is permanent and, if small, occasions little trouble, although there may be diplopia. Pain, some dread of light, and hemorrhage into the anterior chamber are the immediate sequences of such an accident.

Rupture of the sphincter produces mydriasis and minute notchings of the pupil border. According to some authorities the not uncommon dilatation of the pupil (*traumatic mydriasis*) which follows a blow is always accompanied by such a lesion. The condition is not altered by treatment. Rupture of the continuity of the iris membrane by concussion is very rare (Harlan).

Displacement of the iris occurs under three forms: (1) *Retroflexion*, or a folding back of a portion of the iris upon the ciliary processes, usually accompanied by a partial dislocation of the lens; (2) *anteversion*, or turning upon itself of the detached portion of the iris, so that the under or uveal surface is exposed; and (3) *aniridia*, or complete detachment of the iris from its insertion, so that it lies in the anterior chamber, or even under the conjunctiva. An injury severe enough to produce this condition usually is attended with other serious lesions of the remaining structures of the eye.

ANOMALIES OF THE ANTERIOR CHAMBER

1. **Alterations in its Depth.**—Physiologically, the anterior chamber is shallower in infancy and old age, and diminishes in its middle depth during the act of accommodation.

Pathologic **deepening** of the anterior chamber occurs in luxation or absence of the lens, in some cases of cyclitis, and is present in conical cornea and certain forms of staphyloma.

Pathologic **shallowing** of the anterior chamber occurs in chronic iritis with bulging forward of the iris, in glaucoma, and in the later stages of growths of the interior of the eye. Its depth is also lessened when there is diminution of the secretion of aqueous humor, in long-standing inflammation of the uveal tract with detachment of the retina.

2. **Alterations in its Contents.**—These may consist in mere turbidity of the aqueous, as in iritis, keratitis punctata, and glaucoma, or there may be a positive collection of pus, several times referred to under the name of *hypopyon*, and commonly seen in sloughing ulcers of the cornea and purulent inflammations of the iris and ciliary body.

Finally, blood collects in the anterior chamber, a condition which receives the name *hyphemia*. This follows injury to the iris, and occurs in tumors of the eye, hemorrhagic glaucoma, and in severe forms of iritis and cyclitis. It is also seen in hemophilia and splenic leukemia (Sörger). Blood-staining of the cornea may cause a peculiar smoky hue, resembling a lens luxated into the anterior chamber (see also page 411).

3. **Foreign Bodies and Parasites.**—A foreign body penetrating the cornea may lodge upon the iris or fall into the anterior chamber. This may be a fragment of iron or steel or a particle of glass. Sometimes a cilium passing through a wound obtains entrance into the anterior chamber; if it remains long enough, it causes a cystic tumor (*implantation cyst*).

The two parasites described in this situation are *Cysticercus* and *Filaria sanguinis hominis*. In all these instances the intruder should be removed by an operation.

4. **Tumors and Cysts of the Angle of the Anterior Chamber.**—According to Parsons, endothelioma is the only primary growth of the angle of the anterior chamber. A tumor of this character, its cells

Fig. 145.—Cilia in the anterior chamber after wound of corneoscleral junction.

being derived from the pectinate ligament, has been described by Hanke. Tubercle, gumma, sarcoma, and glioma occur as secondary deposits in this region. *Cysts*, derived from a congenital cystic growth of the ciliary epithelium, may develop in the anterior chamber (Holmes Spicer, R. A. Greeves).

CHAPTER X

DISEASES OF THE CILIARY BODY AND SYMPATHETIC IRRITATION AND INFLAMMATION

Cyclitis and Iridocyclitis.—Under the general term *cyclitis* are included various types of inflammation of the ciliary body. The close anatomic connection of the iris, choroid, and ciliary body makes diseases limited strictly to the last structure exceedingly uncommon, just as in many instances inflammations primary in the iris or choroid also involve the ciliary body.

Hence if the iris and ciliary body are associated in pathologic changes, the term *iridocyclitis* is applicable.

The **symptoms** which in general lead to the diagnosis of cyclitis or iridocyclitis are the following: Edema of the lid, injection of the circumcorneal or ciliary zone, neuralgic pain, and tenderness on pressure. Change in the aqueous humor, which grows turbid; precipitates of exudation in grayish-brown points upon the posterior layer of the cornea, and at times hypopyon; exudation in the posterior chamber, attaching the under surface of the iris to the lens-capsule in a complete posterior synechia, the retraction thus produced causing a deepening of the anterior chamber; exudation into the vitreous causing opacities, especially in its anterior layers; and alterations in the tension of the globe, which may be increased or decreased.

The *general symptoms* of pain, photophobia, lacrimation, etc., are present in the acute types of the disease, and vision is seriously impaired according to the amount of the exudation in the pupillary space and vitreous.

To those cases characterized by especially severe ciliary pain and marked pericorneal injection, dilatation of the veins of the iris and decided retraction of its periphery by reason of the plastic nature of the exudate in the ciliary body, the descriptive name *plastic cyclitis* is often given. The tension may be high or low, according to the grade of the inflammation and the character of the process. The disease may involve the choroid, and the vitreous may be filled with opacities. If the pain is comparatively slight and the pericorneal injection less marked, while deepening of the anterior chamber, primary slight dilatation of the pupil, turbidity of the aqueous, and decided precipitation of dots on the posterior surface of the cornea ("keratitis punctata") are conspicuous features, the descriptive name *serous cyclitis* is sometimes applied. With these phenomena fine vitreous opacities, inflammation of the iris and choroid, narrowing of the anterior chamber, increased tension, and secondary glaucoma may be associated.

Purulent cyclitis is characterized by intense ciliary pain, great pericorneal injection, and edema of the conjunctiva and the upper lid. The vitreous contains large opacities, and a noteworthy feature is the formation of hypopyon, which may disappear and reappear again in a few days, its reappearance sometimes being signaled by a fresh exacerbation of intense pain. The iris and choroid commonly are included in the inflammation.

Pathology.—As already noted, systematic writers at one time were accustomed to divide cyclitis into *plastic, serous,* and *purulent* cyclitis. The objections to a classification of this character have been recorded in connection with iritis (see page 306). In general terms, inflammation of the ciliary body may be *acute, suppurative* or *purulent,* and *chronic.* In addition to the infiltration of the iris and exudation in the anterior chamber, there are round-cell infiltration of the ciliary body, much more intense in the vascular ciliary processes than in the ciliary muscle, and lines of exudation into the posterior chamber and the vitreous. The retina, choroid, and nerve are also involved in a varying degree. Later the exudations organize and contract, producing atrophy of the ciliary body, proliferation of the pigment layers, and stretching of the processes toward the posterior pole of the lens. The exudations contain newly formed vessels, the lens becomes cataractous, and if the inflammation has been intense, the retina is detached and *atrophy* of the entire *eyeball* results. If the ultimate result of cyclitis is *phthisis bulbi,* the pathologic process has been a *chronic plastic cyclitis,* with an exudation rich in fibrin which has gradually changed into fibrous tissue.

Prognosis.—Cyclitis under vigorous treatment, begun early, may subside; but the prognosis is always grave, because the disease is liable to originate glaucoma, and in the purulent type, or in the plastic variety which has become purulent, tends to produce atrophy of the iris and choroid and, as described above, *phthisis bulbi.*

Shrunken balls of this character are often tender, readily become inflamed, and may produce sympathetic ophthalmitis; this is particularly true if the original inflammation has been a cyclitis of the plastic type, which probably remains in a *chronic* state.

Causes.—As already stated, primary and uncomplicated disease of the ciliary body is rare. The affection usually is part of a process which involves the choroid or iris, and, therefore, the same conditions and affections which cause iritis (see page 306) may originate cyclitis. A full consideration of the factors concerned with disease of this region will be found on pages 324 and 325, and also in the section devoted to Diseases of the Iris.

Injuries are common causes of cyclitis, and the inflammation may follow operations upon the globe—*e. g.*, cataract extraction.

The **treatment** of cyclitis is practically identical with that of iritis, and, therefore, the directions need not be repeated.

Uveitis, or Serous Cyclitis (*Descemetitis; Aquo-capsulitis; Keratitis Punctata; Serous Iritis*).—In this disease the clinical as well as the pathologic manifestations are chiefly concerned with the uveal tract.

The inflammatory process either affects the whole tract or, first confined to one part of it, is liable to extend to one or both of the other two parts; sometimes it remains confined to the part first attacked.

Causes.—In general terms, the causes of uveitis may be septic or toxic. A certain number of cases depend upon so-called constitutional diseases or disorders of nutrition—for example, chronic rheumatism, gout, arthritis deformans, and diabetes; on specific infectious diseases—influenza, syphilis, gonorrhea, tuberculosis, and specific fevers; on diseases of the blood—for instance, anemia; on auto-intoxications, particularly enterogenous auto-intoxication; on areas of chronic sepsis in the pelvic region, urethra, the prostate and seminal vesicles, the intestines, the rhinopharynx and accessory sinuses, gums (pyorrhœa alveolaris, tooth-root abscess), the tonsils, the pharyngeal ring, and the skin (furuncles). (See also page 314.)

Stock's investigations, both from the clinical and the experimental standpoint, indicate that tuberculosis is a frequent cause of *chronic uveitis*. Hence, tests to determine the presence of tuberculosis should not be neglected in the study of this affection, and the serum reaction of Wassermann should be utilized to establish the presence or absence of syphilis as an etiologic factor. Undoubtedly gonorrhea is a cause of importance. In what manner these various ailments and conditions create an inflammation of the uveal tract has not been positively determined, but it is not unreasonable to assume that the disease manifestations represent an effort on the part of the uveal tract to expel from its tissues some toxin of bacterial or other origin, precisely as certain forms of dermatitis originate in an effort of the skin to eliminate a poisonous agent. Syndey Stephenson maintains that inasmuch as many inflammatory affections of the iris and ciliary body are due to microbic infection, there exist good grounds for believing that the proximate cause of all cases of endogenous iridocyclitis is the excretion by the ciliary body of the micro-organisms and their products. As already pointed out (see page 314), and quoting Stephen Mayou, it may be said that non-suppurative inflammation of the uveal tract is often due to pyogenic organisms of diminished virulence, for example, *Staphylococcus albus*, derived from the distant foci of infection, which have been named.

Symptoms.—In large measure the symptoms of this condition have been described on pages 272 and 315, and in the paragraphs relating to the various types of cyclitis. In other words, the manifestations vary considerably, and one description does not apply to all types.

The following symptoms are often present, and when grouped together are characteristic: There are moderate deepening of the anterior chamber, at the beginning slight dilatation of the pupil (or, at least, an uncontracted pupil), haziness of the cornea and aqueous humor, and a precipitate of opaque dots upon the posterior elastic lamina of the cornea, generally arranged in a triangular manner with the apex pointing upward. There is slight pericorneal injection, and at first no great

tendency to form synechiæ. It is not uncommon to find the tension somewhat higher than normal, at least in the earlier stages of the disease; later it diminishes. With the formation of posterior synechiæ, if they are at all extensive, secondary glaucoma may develop. The one fairly constant clinical sign, which in a certain sense is characteristic,

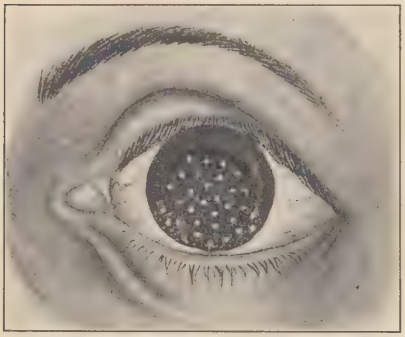


Fig. 146.—Uveitis, early stage; large dots irregularly placed (magnified).

Fig. 147.—Uveitis; large dots on corneal surface (magnified).

is the manifestation which gave rise to the name "punctate keratitis" —namely, a deposit of variously sized and colored dots, arranged usually in a triangular manner on the posterior layer of the cornea. In this connection, however, it should not be forgotten that this so-called descemetitis, in some form or other at least, is practically always present in all varieties of iridocyclitis, although not necessarily in the triangular-like manner which has just been described. The deposits on the posterior surface of the cornea may be fine and dust-like, or large and drop-like. To the last-named variety the term "mutton-fat" is often applied. The deeper layers of the cornea may be infiltrated (keratitis profunda; see also page 303), and the iris may contain thickenings and nodules, especially in the tuberculous types of the disease.

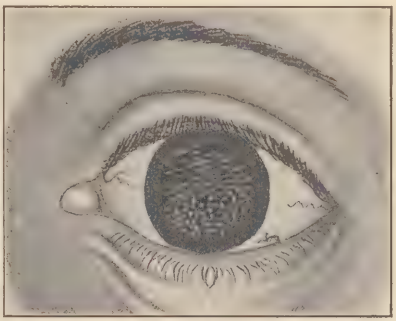


Fig. 148.—Uveitis, showing punctate deposits on cornea and cross-hatching (magnified).

Not only are the evidences of involvement of the iris and ciliary body present, but in most cases careful examination of the choroid will reveal lesions in its tissues. They may be vague, and comprise only the so-called choroidal congestion, or appear far out in the periphery in patches of *acute choroiditis*, sometimes in large zonular areas of *acute plastic choroiditis*, and sometimes so far forward that ophthalmoscopic examination does not reveal their presence. The vitreous usually contains fine, floating opacities, or, in severe cases, coarse,

web-like opacities, and occasionally hemorrhages. The visual field examination will often reveal, even in the absence of rise of tension, irregular contractions, and, not uncommonly in the earlier stages of the disease, *scotomas*. The visual acuteness may be greatly decreased, owing to exudations in the vitreous and the deposits on the cornea, while in some of the milder cases it is scarcely reduced below the normal, and the patients are conscious only of ocular disease because of slight local discomfort and muscæ in their field of vision.

There are so many manifestations of this disease that it is not practicable to attempt an exact classification. In general terms, it may be said that sometimes the signs are chiefly those of punctate keratitis or descemetitis; that a senile form of the disease is not uncommon, unassociated with acute symptoms or involvement of the iris, with only a few spots on the posterior surface of the cornea, and with a certain amount of flaky vitreous opacity, choroiditis in active manifestation being absent, although there may be a certain amount of irregularity of the retinal circulation; that occasionally a marked descemetitis is the chief sign of a decided choroiditis or choroido-retinitis, often of obscure origin; that a chronic uveitis may develop in the iris, less commonly in the ciliary body, with or without punctate deposits in the cornea, insidious in character, more frequently encountered in young women than in men or in children; and that, finally, the disease may be recurrent and assume a type to which the author has ventured to give the name *malignant uveitis*, and which terminates in secondary glaucoma, cataract, and often in blindness.

With these cases of malignant uveitis may be described those to which the names *iridochoroiditis, cyclitis with disease of the vitreous and keratitis punctata*, and *chronic serous iridochoroiditis* have been given. They have been divided into two forms, according as the affection is primary in the iris or in the choroid. In the first instance, there are mild iritis, insignificant pain, and ciliary congestion, deepening of the anterior chamber, and spots on the posterior layer of the cornea; inflammation continues, relapses take place, exudation occurs behind the iris, while its pupillary margin is bound down so that the surface is irregularly bulged forward, and if the pupil is not too much occluded, the ophthalmoscope will reveal many flocculi in the vitreous. The tension may now rise and the eye pass into secondary glaucoma.

In the other type the process passes from behind forward, beginning with patches of choroiditis, which increase in extent and depth; the nutrition of the vitreous is impaired and opacities form, the lens is altered and pushed forward, the iris becomes embodied in a plastic inflammation, with narrowing of the anterior chamber and a loss of vision. As the disease of the uveal tract continues, the lens becomes opaque, the eyeball softens, the retina may be detached, and, finally, phthisis bulbi occurs. In addition to the causes already mentioned, the affection, which is common in young adults, and usually symmetric, has been attributed to prolonged work, associated with loss of sleep and defective nutrition.

Pathology.—The deposits on the posterior surface of the cornea, which are so conspicuous a symptom of this disease, and which have given rise to the name punctate keratitis, may be very fine or of medium size, and sometimes large and greasy-looking, in which circumstances they are known in England as "mutton-fat deposits." They are derived chiefly from the ciliary body, as Fuchs originally demonstrated, and also, in lesser degree, from the iris. An exudation of lymphocytes occurs upon the surface of the ciliary body, the lymphocytes pass into the anterior chamber and are precipitated on the posterior surface of the cornea (Fuchs). Their cyclitic origin has been strongly maintained by E. Treacher Collins, especially after his discovery of the so-called glands of the ciliary body. In other words, according to him, this disease may be regarded as primarily a catarrhal inflammation of these glands. Their secretion, he maintains, becomes augmented, causing increase in the aqueous humor and deepening of the anterior chamber. The aqueous is altered in character, contains leukocytes, pigment cells, and fibrin, and these formed elements gravitate and are deposited upon the lower portion of the posterior face of the cornea. Some authors—for example, Hill Griffith—have asserted that the dots on Descemet's membrane are formed in the choroid and set free in the vitreous, are carried by the nutrient currents of the eye and deposited on the back of the cornea. His view, as he himself points out, would necessitate the admission that the suspensory ligament is permeable to solid particles. Histologic investigation of the eyes affected with this disease indicate that in general terms there is a chronic cyclitis, in which the anatomic changes do not materially differ from those found in ordinary types of cyclitis. Groenouw has demonstrated round-celled infiltration of the iris, of the deeper layers of the corneal border and the ciliary body, with collections of round cells on the posterior layer of the cornea and on the ciliary processes. In his investigations the choroid, retina, and optic nerve were normal, but other observers have found them affected, as necessarily would be the case in so far as the choroid is concerned, where the ophthalmoscopic evidences of choroiditis are present. In later stages of chronic uveitis there is an overgrowth of the epithelium of the ciliary body. This epithelial hyperplasia, described by Fuchs, is, according to him, characteristic of some cases of chronic uveitis. Harms' investigation of the pathologic anatomy of chronic iridocyclitis with deposits on Descemet's membrane indicates that the inflammation of the uveal tract is limited largely to the anterior segment of the eyeball, the deeper parts being much less affected. The pigment found in the deposits on the posterior surface of the cornea arises probably in part from degenerated red corpuscles, and largely from the uveal tract. In some grave types of iridocyclitis, leading to retinal detachment and atrophy of the eyeball, Fuchs found lesions resembling exogenous *endophthalmitis*, and believes they are caused by a virulent toxin or by bacteria conveyed by metastasis.

Treatment.—This depends upon the character of the disease or the type which it assumes. In the absence of increased intra-ocular

tension, mydriatics are indicated, either atropin or scopolamin. With increased tension the mydriatic must be suspended and occasionally a miotic may be needed, or paracentesis of the anterior chamber may be performed. Dionin is of distinct advantage, and it may be combined with the atropin or eserin, according to indications. Pilocarpin diaphoresis is sometimes admirable in its effects. If for any reason the drug is not well borne, a similar physiologic action may be obtained by sweats induced in a Turkish bath, or with the aid of an ordinary cabinet. Subconjunctival injections are exceedingly valuable —either ordinary physiologic salt solution or cyanid of mercury. Mercury, preferably by inunction, even in non-specific cases, should be exhibited. It may also be given by the mouth in the form of the protiodid. In syphilitic cases the indications for mercury are evident, and *salvarsan* and *neosalvarsan* (see page 310) should be employed. Iodid of potassium, iodid of sodium, and iodid of lithium are important alterative remedies, and in acute cases, especially those associated with great pain and decided cyclitis, full doses of salicylate of sodium or of aspirin render signal service. Naturally, the indications furnished by the probable etiologic factors must be given due consideration, and, therefore, iron, arsenic, bichlorid of mercury, syrup of hydriodic acid, and similar remedies should be administered. With atoxyl in this disease the author has had no experience, but he has seen marked benefit follow the administration of Donovan's solution. The administration of tuberculin in the manner already described (see page 318) is followed by excellent results, and should certainly have full trial if the reaction to tuberculin is positive. Mayou has successfully treated uveitis with *staphylococcic vaccine* where the staphylococcus has been obtained from the anterior chamber by paracentesis, and tests have demonstrated that neither tuberculosis nor syphilis is the etiologic factor. The author's investigations indicate that in a certain number of cases of uveitis, especially of an insidious and relapsing type, so-called enterogenous auto-intoxication bears a relation to the disease, even if it is not a causal one in the sense that a toxin formed within the metabolism is the exciting agent, and treatment should include a carefully selected diet and intestinal antisepsis. Indeed, the association of intestinal sepsis with this disease is often an intimate one. The important relation of focal infections in the rhinopharynx, the teeth, and the accessory sinuses to this affection has been referred to, and these regions should be thoroughly explored and treatment ordered according to the findings. The investigation of the teeth should include an *x*-ray examination. As Allen J. Smith and Barrett have found that the *Endameba buccalis* is one of the factors in the production of pyorrhœa alveolaris, emetin has been used with success in relieving the oral sepsis, and thus indirectly may help in the cure of a uveitis caused by it. After the acute symptoms have subsided, in the opinion of some authors massage of the eyeball is desirable, and galvanism has been advocated. S. Lewis Ziegler believes that electric treatment shortens the course of uveitis. He advises the application of the positive pole. In some types of

malignant uveitis the administration of thyroid extract has achieved encouraging results (Bordley).

If as the result of this disease firm posterior synechiæ block the communication between the anterior and posterior chambers, this should be reopened by a broad peripheral iridectomy, which, if the lens is opaque, may be combined with its extraction. Even in eyes in which softening has begun, provided the field of vision still remains intact, good results will sometimes follow a successful iridectomy. If at any time during the course of the disease rise of tension should develop and iridectomy should not be admissible, either paracentesis of the anterior chamber or posterior sclerotomy may be tried. Tapping the anterior chamber has been advised as a therapeutic measure, even if the tension is not elevated, and in a few cases in which the author has put this expedient to the test it has apparently been of benefit to the patients. The fluid from the chamber should be examined for the *Spirochæta pallida*, tubercle bacilli, and for pyogenic organisms. From the latter vaccines can be prepared if the treatment advocated by Mayou is to be tried.

In recent times a few reports indicate that *radium* has served a useful purpose in relieving the pain and facilitating the absorption of inflammatory products in chronic uveitis (C. H. Williams).

Injuries of the Ciliary Body.—The danger attending perforating wounds of the sclera has been described on page 292; this danger is doubly increased if the wound occurs in any portion of a zone, $\frac{1}{4}$ inch wide, surrounding the cornea, a region commonly called the "dangerous zone," after the late Mr. Nettleship's apt description. In addition to the damage inflicted by the wound the risk of acute and suppurative cyclitis and of sympathetic inflammation is present.

Treatment.—After a penetrating wound in this region two courses are open to the surgeon—an attempt to save the eye, or immediate enucleation. If an attempt be made on the side of conservatism, the plan discussed under scleral wounds (see page 293) should be followed; if not, enucleation or one of its substitutes will be required (see page 636).

Syphilis of the Ciliary Body.—Syphilitic affections of the ciliary body may exist either in a diffuse infiltration of granulation tissue and inflammatory exudation or in tumor formation—that is, in *syphiloma of the ciliary body*, to adopt the term advised by Ewetzky, who has thoroughly studied this subject. According to him, the symptoms of syphiloma of the ciliary body manifest themselves chiefly in the form of a severe iridocyclitis, with hazy cornea, or an intense parenchymatous vascular opacity of this structure. Keratitis punctata may be a prominent symptom, and sometimes hypopyon is present. The tumor formation in the ciliary region may extend completely around the cornea, and perforation usually takes place through the sclerotic or into the anterior chamber. The color of the tumor is often yellow, and when caseous degeneration sets in there may appear in the anterior chamber the products of the degeneration of the tumor, which give rise to the appearance of hypopyon. The largest number of cases occur

between the twentieth and the fortieth years of life, and more men than women are affected. Only rarely is inherited syphilis a cause of this condition. In those cases due to acquired syphilis, a large percentage has appeared in the early stage of the systemic affection.

Fig. 149.—Gumma of iris and ciliary body.

A number of cases of *gumma of the ciliary body* have been recorded, but on pathologic grounds it is probably impossible to distinguish between papules and gummas, and, therefore, Ewetzky's term *syphiloma* is appropriate. Some writers describe *precocious gummas of the ciliary body* as early tertiary lesions, but Ewetzky does not believe that they should be separated into a special group. The condition must be differentiated from syphilitic growths of the conjunctiva which are movable; from syphiloma of the sclerotic, which is usually unassociated with iridocyclitis, and from melanotic sarcoma and tuberculosis of this region. The *treatment* does not differ from that which has been advised in connection with syphilitic diseases of the iris, and full doses

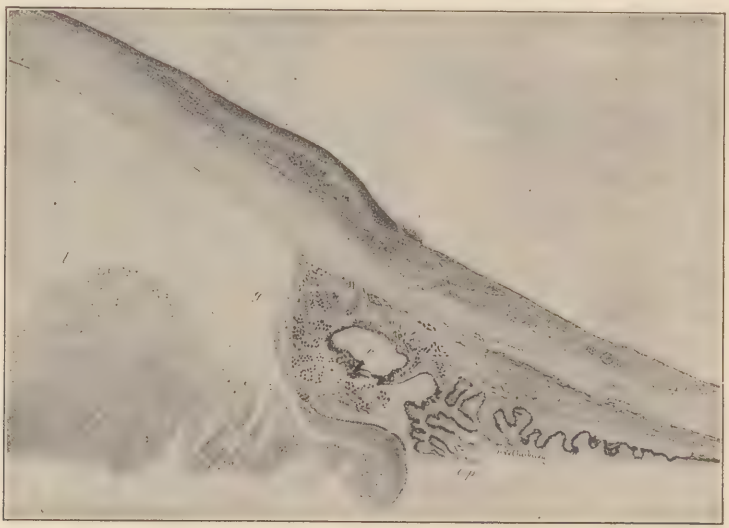


Fig. 150.—Microscopic section of gumma of iris and ciliary body (see Fig. 149): *l*, Remains of lens; *c. p.*, atrophied ciliary processes; *g*, gummatous growth involving base of iris and ciliary body, containing in its center a cyst, *c*.

of mercury and iodid of potassium are the most important therapeutic agents, especially if used in conjunction with salvarsan and neosalvarsan (see page 310).

Tumors of the Ciliary Body.—Primary sarcoma of the ciliary body is a rare disease. Usually the growth is pigmented, although a few cases of leukosarcoma have been described. The sarcoma may not seriously impair the function of the eye in its earlier stages, when the tumor appears as a brown mass behind the iris, rarely in the angle of the anterior chamber. Later the growth exhibits the four stages which are common to all intra-ocular tumors. The tumor may be composed of round, spindle, or mixed cells, and, according to Groenouw, the prognosis is better than in sarcoma of the choroid. Sometimes sarcoma of the ciliary body assumes a flat and infiltrated character, to which the name *ring* or *annular sarcoma* has been given. This is a comparatively rare manifestation, only about 8 cases being on record (Alling and A. Knapp). The ciliary body may also be invaded by sarcoma from the choroid or iris.

Other growths which have been noted in this region are *epithelial hyperplasias* and *innocent tumors* of the *ciliary epithelium* (Fuchs), which, occurring in elderly persons, arise from the unpigmented layer at the summit of a ciliary process. An unusual pathologic condition found by Fuchs in an eyeball atrophic after iridocyclitis is a *neuroma of a ciliary nerve*. *Adenomas* have been described and, very rarely, *primary carcinoma*, *myoma*, and *myosarcoma*. For those tumors of the ciliary epithelium which represent the embryonic retina Fuchs proposes the name *diktyoma*. A tumor of this character has been described by Verhoeff with the title *teratoneuroma embryonale*. *Metastatic carcinoma* and *secondary glioma* have been recorded.

As an extension of the disease from the iris or choroid, *tubercle of the ciliary body* may appear, and *leprosy nodules* have been reported, as have also a few *cysts*.

Senile degeneration of the ciliary body occurs, and *atrophic changes* in this structure are met with after cyclitis. According to Parsons, *calcification* and *ossification* of the ciliary body itself are rare. Nearly always there are sequels of ossification in the choroid.

SYMPATHETIC IRRITATION AND SYMPATHETIC INFLAMMATION OR OPHTHALMITIS

These terms are applied to affections in which one eye is implicated in a morbid process as the result of disease or injury to the other. For a long time it was taught that they represented two essentially different conditions; but in recent years a number of authorities have concluded that this distinction should no longer be maintained—that, in other words, sympathetic irritation should be regarded simply a moderate degree of sympathetic inflammation. Schirmer, however, continues to insist on the essential difference of the two affections, and, on clinical grounds at least, and in the light of present knowledge, it seems wise to agree with his contention.

It is customary to describe the eye which is implicated as the result of disease or injury of its fellow as the *sympathizing eye*, and the one

affected by the disease or injury which causes the irritation or inflammation the *exciting eye.*

Conditions Producing Sympathetic Affections.—Generally one or other of the following conditions is present:

(1) Wounds of the ciliary region which set up a traumatic iridocyclitis or uveitis, especially if associated with incarceration of the underlying tissue in the scar, as well as wounds of the eyeball elsewhere located. The ciliary region is the zone previously described by the term borrowed from Mr. Nettleship, "dangerous zone." Traumatisms probably cause over 80 per cent. of the cases of sympathetic inflammation. (2) Foreign bodies in the eye. (3) Perforating wounds or ulcers of the cornea in which the iris has become incarcerated, or scars involving the ciliary body. (4) Operations upon the eye—extraction of cataract, sclerotomy, iridodesis, iridectomy, discission, and reclination. (5) Luxation, wounds, and calcification of the lens. (6) Intra-ocular tumors, *e. g.*, sarcomas (Fuchs, Meller), especially if associated with iridocyclitis (Schirmer). (7) Ossification and calcification of the choroid and ciliary body. (8) Pressure of an artificial eye or incarceration of the stump of the optic nerve in scar tissue, after the operation of enucleation, and, in rare instances, implantation of an artificial vitreous in Tenon's capsule or in the scleral cup (Mules' operation).

According to O. Schirmer, there is no sound evidence that herpes zoster ophthalmicus, glaucoma, symblepharon, intra-ocular cysticercus, subconjunctival rupture of the globe (without associated iridocyclitis), or spontaneous inflammation of one eye can cause sympathetic ophthalmitis, although sympathetic irritation may, no doubt, arise in consequence of any of these conditions. Meller has reported some cases of sympathetic ophthalmia, the exciting eye having acquired inflammation spontaneously, that is, without injury, but in each instance iridectomy had been performed on the eye originally attacked. Whether eyes which are, or have been, the subjects of purulent panophthalmitis can produce sympathetic inflammation is disputed. This relationship has been recorded (Alt,[1] Schirmer, Ahlström, Zentmayer). According to Ruge, even in these eyes the real exciting cause was a fibrinoplastic inflammation.

Sympathetic irritation, according to some authors, should be regarded as a functional disturbance—that is, a *sympathetic neurosis.* It presents a series of symptoms, comprising photophobia, lacrimation, blepharospasm, defective or impaired accommodation, lessened visual acuteness or amblyopia, inability to perform close work, neuralgic pain through the distribution of the supra-orbital nerve, tenderness on pressure over the ciliary region, photopsia, contraction of the field of vision (fatigued visual field), and hyperemia of the eye-ground.

[1] It is stated that eyes which are, or have been, the subjects of purulent panophthalmitis do not excite sympathetic ophthalmitis, and generally in suppuration of the cornea and its sequels and in panophthalmitis and the phthisis bulbi which it causes, this complication is not to be apprehended (Fuchs). Alt, however, in his analysis of more than 100 cases, found 13 eyes enucleated for sympathetic iridochoroiditis, the other having been lost by purulent panophthalmitis.

The tendency of this condition is to recur, and in this sense it may last for weeks, or even for months and years. It disappears entirely with the removal of the exciting eye or of the exciting cause. Those authors who decline to regard this affection as one which should be separated from sympathetic inflammation believe that its origin is infective, and that the character of its manifestations is due to a small or intermittent dose of the toxin.

Symptoms in the Eye Exciting Sympathetic Irritation.—An eye so injured or diseased that it is liable to produce the condition described in the preceding paragraph is liable, during the course of the irritation, to attacks of congestion in the ciliary region, photophobia, tenderness on pressure, lacrimation, and neuralgic pain. Sympathetic irritation may also be induced by minor lesions—for example, retained foreign bodies in the conjunctival cul-de-sac.

Sympathetic inflammation (*sympathetic ophthalmitis; infective cyclitis, transferred ophthalmitis* [Oliver]) occurs in several forms, sometimes arising in the wake of an attack of irritation, sometimes coexisting with it, but frequently without any premonition or association of this character. It has been stated that marked oscillation of the iris often occurs when a sympathetic irritation is about to give place to an inflammation. H. S. Gradle believes that marked *lymphocytosis* is present if the injured eye is of such a character that it may produce sympathetic ophthalmia. The diagnostic and prognostic value of the test is disputed by Neumann and Franke.

With or without warning, sympathetic ophthalmitis or, as it may be called, *sympathetic* or *infective uveitis*, because the uveal tract is especially involved, presents itself:

1. As an *iridocyclitis* (*uveitis fibrinosa sympathetica*, Schirmer), plastic or malignant—*i. e.*, an inflammation characterized by pain, photophobia, pericorneal congestion, discoloration of the iris, closure of the pupil by exudation around its margin and behind the iris, which is plastered to the capsule of the lens, cyclitis, narrowing of the anterior chamber, effusion into the vitreous, involvement of the choroid, opacity of the lens, detachment of the retina, and finally shrinking of the eyeball.

According to Schirmer, ophthalmoscopic changes are frequent in the early stages of sympathetic uveitis, in the form of a choroidoretinitis, the outlines of the papilla being hazy, the retina edematous, and the retinal veins dilated and tortuous.

2. As a *serous iritis*, more accurately a *serous iridocyclitis* (Panas), or *serous iridochoroiditis* (de Wecker), or *serous sympathetic uveitis* (Schirmer), causing turbidity of the aqueous, deepening of the anterior chamber, punctate opacities on the posterior layer of the cornea, slight rise in tension, moderate ciliary injection, opacity in the anterior layers of the vitreous, some involvement of the ciliary body and choroid. Not infrequently, if not in all the cases, *papilloretinitis* coexists with the uveitis. This process under proper treatment may cease and recovery result; but often it may pass into, or be the forerunner of, a malignant uveitis, with all its evil consequences.

3. As a *papilloretinitis* (*sympathetic papilloretinitis*, Schirmer), which as a coexisting condition in uveitis has been noted. Sometimes, however, according to Schirmer, it constitutes the primary affection; that is, the uvea is not associated in the inflammation. This neuritis is of moderate grade, the disk is not prominent, its borders are veiled and surrounded by grayish retinal opacity; the veins are swollen and tortuous, and occasionally small hemorrhages are present.

A *choroiditis* caused by sympathetic inflammation has been described a number of times, notably by Hirschberg, Caspar, Haab, and A. Dalén. The lesions somewhat resemble the spots of disseminated choroiditis due to syphilis, and appear, especially in the periphery of the eye-ground, in the form of small yellowish-red areas, with central pigment dots. According to Dalén, the disease is a chorioretinitis, and not a pure choroiditis. A peculiar distribution of small yellowish-white flecks or nodes, lying behind the retinal vessels, often grouped and without pigmentation, has been described; they probably lie within the choroid. Choroiditis in the primarily affected eye has been recorded (Hirschberg, Heerfordt).

4. Other manifestations of sympathetic ophthalmitis have been reported, for example, a *simple atrophy of the optic nerve*, but the exact relationship of such a condition to a sympathetic affection has not been proved. In this connection reference should be made to the so-called *sympathetic amblyopia* described by Nuel, which, according to this author, begins at a much later period than true sympathetic ophthalmia —that is, at a period later than one or two months after the traumatic iridocyclitis has occurred. At first there is only a vague amblyopia, with obscurations; later visual acuteness is much reduced and the field of vision contracted, and should the amblyopia attain a decided degree, there may be a slight neuritis or pallor of the papillomacular bundle, or a perivasculitis. The affection may continue for months or even years, with alternate improvements and aggravations. Nuel explains it by assuming the presence of a neuritis caused by a hyperplasia of the interstitial tissues. Deafness has been observed.

There is no good evidence to show that cataract, conjunctivitis, keratitis, or scleritis are manifestations of that affection to which the name "sympathetic" is ordinarily applied.

A premonitory symptom of great importance, and one which should always be searched for in cases in which sympathetic irritation or inflammation is likely to take place, is an almost characteristic tenderness in the ciliary region, frequently in a circumscribed spot, which may be picked out with the end of a probe. When this is pressed upon, the patient shrinks from the touch in a peculiar and striking manner. Sometimes an exactly similar tender spot is found in the ciliary region of the exciting eye. Biehler and von Hippel have demonstrated that fluorescein will color the endothelium of the cornea in certain uveitic inflammations when the superficial layers of the cornea are still intact and when ordinary examination fails to reveal these early changes. Alberti suggests the use of this test in cases of suspected but not yet

manifest sympathetic inflammation. A. Maitland Ramsay and A. W. M. Sutherland have observed, using Bjerrum's method (see page 81), spindle-shaped enlargement of the blind-spot as a sign of active congestion of the optic disk, and suggest this examination as an important aid in determining the onset of trouble passing from an eye with an infected injury to the fellow eye. Von Hippel has applied Abderhalden's test in cases of sympathetic ophthalmia; in these patients a positive biologic reaction to uveal tissue was obtained, but the reaction is not specific for sympathetic ophthalmitis.

In view of the prominence which the metastasis-theory of sympathetic ophthalmitis has attained in recent times, and to which reference is made on page 338, the *general symptoms* of this condition are of importance. The presence of meningitis has been suspected, but never demonstrated. Severe headache, however, rise of temperature, delirium, and deafness have been reported, and it is of the utmost importance to submit patients who are suffering from sympathetic ophthalmitis to the fullest investigation from the general standpoint. Römer and the author have urged that the blood of patients with sympathetic ophthalmia should be carefully examined for micro-organisms. Lymphocytosis, as described by Gradle, has been referred to.

Symptoms in the Eye Exciting Sympathetic Ophthalmitis.—Preceding the development of any of the types of sympathetic ophthalmitis, the exciting eye usually presents obvious iritis or iridocyclitis, congestion, and alteration in the tension; but the local manifestations in the exciting eye may not be characterized by pain, and consequently may escape attention, and although necessarily the vision is disturbed, the eye need not be a blind one. Indeed, there are no phenomena in the exciting eye which may be designated as characteristic, and Schweigger was unwilling to recognize a clinical picture peculiar to the sympathizing eye.

The **period of incubation,** or that period of time between the reception of the injury or disease in the exciting eye and the development of *inflammation* in the sympathizing eye, varies considerably, in the majority of cases being from three to six weeks. Exceptionally the disease begins as early as the fourteenth day and has been postponed as late as twenty years, in Alt's collection one case being stated to have occurred as late as sixty years after the exciting disease. The maximum interval is, however, difficult to state with accuracy.

Sympathetic irritation may arise within a very few days after the reception of an injury. It has occurred within the first forty-eight hours.

Pathology.—In so far as sympathetic irritation is concerned, if this is to be considered as a condition distinct from sympathetic inflammation, no information in regard to pathologic changes can be given, because, to all intents and purposes, they do not exist, or, if they exist, opportunity to detect them has not been afforded. It is usually considered to represent a neurosis, and the exciting eye to have conveyed through the medium of the ciliary nerves the irritation-phenomena which are manifest in the sympathizing eye. It is to be noted, however, that the

exciting eye, in these circumstances, may present exactly the pathologic changes which in other circumstances produce a true ophthalmitis in the fellow eye, and, therefore, the belief of many authors that the two are identical is not to be disregarded.

The pathologic anatomy of eyes which produce sympathetic ophthalmitis, that is to say, exciting eyes, has been thoroughly investigated, while that of the eyes which have become inflamed as the result of an infective cyclitis of the fellow eye, that is, sympathizing eyes, has not received much attention, for the simple reason that the opportunities of examination are rare. In so far, however, as they have gone, in general terms it may be stated that the lesions are identical; in other words, the same conditions are present in the exciting and the sympathizing eye.

Fig. 151.—Traumatic iridocyclitis. Diffuse infiltration of the iris and ciliary body.

Schirmer has summarized the lesions in uveitis which produce sympathetic ophthalmitis somewhat as follows: All three portions of the uvea contain disseminated foci of small round cells, associated with a high grade of inflammation, characterized by a diffuse infiltration of the entire tissue with similar small cells. After the disappearance of the inflammation the uveal structure is destroyed and atrophied, and substituted by a pigmented connective tissue poor in vessels. Upon the surface of the iris and ciliary body there is a rich fibrinous exudation, with a strong tendency to organization, but the choroid fails to exhibit a similar exudative process.

Fuchs' investigations have thrown a new light on the pathologic anatomy of sympathetic ophthalmitis. He found in the uvea of exciting eyes a dense infiltration of lymphocytes, and in many cases in the midst of this infiltration collections of epithelioid cells, often with giant

cells between them. This infiltration may be present only in certain spots in the form of isolated nodules in the iris, ciliary body, or choroid, or the uvea may be wholly or at least largely occupied by the process. Indeed, the infiltration may make its way into the sclera, which becomes permeated with scattered nodules. In so far as the different tissues of the eye are concerned and their implication in this process, the iris is the least affected, the ciliary body is always involved, and usually the choroid is more infiltrated than other portions of the uvea, especially in its posterior portion. A fibrinoplastic uveitis not infrequently complicates this *proliferative uveitis* of sympathetic inflammation, but Fuchs does not regard this as an essential factor in the process, because it may be absent in typical cases. Bacteria were not demonstrated in the nodules, but Fuchs does not doubt that the affection is produced by bacteria, which, instead of causing an acute suppuration, originate a chronic proliferation, and this inflammation has the property of being transmitted to the second eye through the circulation.

Fuchs' observations have been confirmed by Lenz, Kitamura, E. V. L. Brown, and a number of other observers. Ruge, however, denies that the anatomic conditions found in an eye exciting sympathetic ophthalmitis are characteristic, and while he does not doubt that there is a mixed infection in many cases, he holds to Schirmer's view that there is a pure sympathetic plastic exudation. Meller concludes that such marked specific changes cannot always be found in the exciting eye as would justify a diagnosis, but such insufficiency of histologic findings does not alter the view that there is in the first eye a specific morbid process similar to that by which the ophthalmia of the second eye is produced and which is distinctly different from posttraumatic inflammations. Now, while it may be too much to say that proliferative uveitis is pathognomonic of sympathetic ophthalmitis, there seems no doubt, to use the language of E. V. L. Brown, that it is the essential anatomic condition present in the eye which causes sympathetic inflammation of its fellow.

Pathogenesis of Sympathetic Ophthalmitis.—Formerly it was almost universally thought that this disease was due to a reflex action through the ciliary nerves, and on this theory the name "sympathetic" was applied. The exact nature of this grave malady is not known, although the older hypotheses have largely been abandoned for the *theory of infection.*

According to Deutschmann, the inflammation is a progressive process in the continuity of the tissue of one eye to the other by way of the optic nerve apparatus, and is of bacterial origin; hence, a *migratory ophthalmitis.* Deutschmann's researches have, however, not been confirmed (Gifford, Mazza, Randolph, Limbourg, Levy, and Greeff), and "the migration theory is still a hypothesis" (Greeff). The ciliary nerve theory, in a modified form—*i. e.*, that disturbances of nutrition and in the circulation, caused by irritation of the ciliary nerves in the "exciter," create the inflammation in the "sympathizer"—has been maintained by Schmidt-Rimpler. Römer's investigations, however, have utterly

set aside the possibility of accepting the ciliary nerve theory in any form. Bellarminoff and Selenkowsky believe that all cases of sympathetic disease may be explained by the action of a toxin which is produced by the bacteria which have entered the primarily affected eye, and which reach the other eye by means of the lymph and diffusion streams. Brown Pusey suggests as an explanation of sympathetic ophthalmitis that the cells of the injured eye, probably those of the ciliary body and iris, give rise to a cytotoxin, which, having a selective affinity for corresponding cells in the other eye, there sets up an inflammation. A precisely similar theory has been propounded by Golovine. Motais, although frankly stating that he has no clinical evidence to support him, and that as yet no experimental researches are at hand to lend aid to his hypothesis, maintains that the anastomoses between the veins of exit of the eyes constitute the most probable paths for the transmission of sympathetic ophthalmitis; and even so great an authority as Leber believes that this view should receive consideration. Römer regards sympathetic ophthalmitis as a *metastasis*, the metastatic infection proceeding by way of the blood-streams, and thus brings himself in accord with views originally expressed by Berlin. This infection is ascribed to some form of micro-organism which is pathogenic for the eye alone and does not affect the body generally. The metastasis theory, according to Römer, most satisfactorily explains all of the phenomena of so-called sympathetic ophthalmitis, and is entirely consistent with the results of modern bacteriologic research. Moreover, it has now received confirmation from the anatomic standpoint, especially by Fuchs' researches.

The micro-organisms which excite sympathetic ophthalmitis have not been isolated; indeed, they have not been seen, and, therefore, it may be assumed that they are distributed in invisible form by the intraocular fluids throughout the eye, and the condition represents, as Parsons puts it, a generalized endogenous infection by invisible organisms. Attempts have been made by Raehlmann with the "ultramicroscope" to detect these bodies, and, according to his report, he has met with success. It would seem, however, that his researches require considerable confirmation, and Zur Nedden's rod-like bacillus, which "seemed to fulfil the conditions demanded of the exciter of sympathetic ophthalmia," has not been proved to be the potent agent in this respect.

While the prevailing theory is that in sympathetic ophthalmia the infection enters the eye through a wound or other pathway, J. Meller has advanced an *endogenous theory*. According to him some part of the body other than the eye is the port of entry for the specific organism which has an elective affinity for uveal tissue, and attacks that of the diseased eye because its resistance is lowered, the damage having occurred from a wound or a toxic iridocyclitis. Gradually sympathetic inflammation arises, which leads to metastasis to the other eye. E. V. L. Brown believes that this theory explains those cases in which in the primary eye neither wound nor other opening can be found.

Recently Elschnig has maintained that in the development of sympathetic ophthalmia two factors are necessary: *anaphylaxis* of uvea, on account of tissue disintegration, the chief rôle being taken by the pigment, and an anomalous condition of the organism, for example, nephritis, diabetes, auto-intoxication, etc. Naturally, this theory of the existence of *anaphylactic uveitis* has met with much opposition, but recently has acquired some advocates, for instance, A. Jess, who admits it explains certain clinical facts better than the bacterial hypothesis, to wit, the period of incubation, and Cramer, who observed a second attack of sympathetic ophthalmia coincident with alopecia and whitening of the eyebrows. This he believed was an anaphylatic reaction of pigment origin.

Treatment.—The most important consideration is *prophylaxis*, or, in other words, the management of the eye originally affected. This depends upon the character and situation of the wound or upon the stage of the disease, and upon the amount of vision possessed by the injured or diseased organ.

In the section devoted to Treatment of Wounds of the Sclera (see page 293) the method is described by which eyes seriously wounded may be saved. Schirmer believes that the treatment of injured eyes should include full doses of mercury, and the author can confirm the therapeutic value of this remedy in this respect, as well as the value of large doses of salicylate of sodium.

It may sometimes happen, where every advantage of nursing and careful watching is at hand, that eyes may be saved which would be sacrificed in other circumstances. The attempt requires the gravest thought before it is undertaken, because the onset of sympathetic ophthalmitis may be insidious, and when once begun treatment rarely fully removes the structural changes which have taken place. The propriety of operating must be determined by regarding the following rules, which are modified from those given by Schirmer and Swanzy, and represent the published experiences of the best authorities.

Enucleation, or one of its substitutes, should be performed on—

1. An eye with a wound so situated as to involve the ciliary region, and so extensive as to destroy sight immediately, or to make its ultimate destruction by inflammation of the iris and ciliary body reasonably certain.

2. An eye with a wound in this region already complicated by severe inflammation of the iris or ciliary body, even if sight is not destroyed; or an eye containing a foreign body which judicious efforts have failed to extract, and in which severe iritis is present, even if sight is not destroyed.

3. An eye the vision of which has been destroyed by plastic iridocyclitis, or one which has atrophied or shrunken, provided there are tenderness on pressure in the ciliary region and attacks of recurring irritation; or without waiting for signs of irritation.

4. An eye the sight of which has been destroyed, even though sympathetic inflammation has begun in the sympathizing eye, in the hope of

removing a source of irritation, and thus rendering treatment to the second eye more effectual.

5. An eye in which the wound has involved the cornea, iris, or ciliary region, either with or without injury to the lens, and in which persistent sympathetic irritation in the fellow eye has occurred, or in which there have been repeated relapses of sympathetic irritation.

6. An eye either primarily lost by injury or in a state of atrophy, associated with signs of sympathetic irritation in the fellow eye.

It is universally conceded that the enucleation of an eye (*preventive enucleation*) primarily injured, the visual function of which cannot be restored, is the surest way of preventing sympathetic ophthalmitis. It is to be remembered, however, that even a very early enucleation does not necessarily prevent sympathy in the fellow eye, because the infective process may have begun before the operation, and may not develop for several weeks. It has occurred fifty-three days after the enucleation of an eye injured twenty days prior to its removal (Stephenson), and C. B. Welton has collected 28 cases of this character, the shortest interval between the injury and the enucleation being nine days and the longest two months. In place of enucleation, evisceration has been practised, but this operation has been followed by sympathetic inflammation. Resection of the optic nerve (neurectomy) does not provide absolute security, but if the patient declines enucleation, it may be used as a substitute.

If sympathetic inflammation has begun, the rules just quoted are not applicable, and *enucleation must not be performed if there is any vision in he exciting eye*, which in the end may prove to be the more useful organ. The treatment already recommended for iritis and iridocyclitis is applicable.

In the treatment of the sympathetically affected eye operation usually has no place. Both iridectomy and sclerotomy have been advised, but it is better to await the subsidence of acute symptoms before attempting any surgical interference unless the intra-ocular tension is inordinately raised, when scleral incision may be practised or iridectomy with or without the removal of the lens.

The *general treatment* consists in confinement in a darkened room (moderate exercise with eyes well protected is permissible in subjects failing for lack of it); complete functional rest of the eyes and atropin or scopolamin locally, provided there is no rise of tension and no atropin irritation; and leeches to the temple if the inflammation is florid. Dionin (5 per cent.) should be used. Mercurial inunctions are important, and free diaphoresis, either with pilocarpin or by vapor baths, has been advised; in debilitated patients tonics and alteratives are advisable. The value of sodium salicylate, advocated by Gifford in the treatment of sympathetic ophthalmitis, is great, at least 60 to 100 grains (3.9–6.5 gm.) a day should be exhibited, and even larger doses are sometimes well borne; but, inasmuch as the treatment must continue for long periods of time, the doses must be regulated strictly according to the results achieved. Gifford believes that most patients will be able to take daily

1 grain (0.065 gm.) of sodium salicylate for each pound of weight. While this drug cannot prevent sympathetic uveitis, its use during traumatic iridocyclitis seems to render the sympathetic affection less virulent than otherwise would be the case, and in this respect is the superior of mercury. Stock recommends benzosalin in the treatment of sympathetic ophthalmia, and Gifford has found atoxyl to be useful, and recently has advised large doses of atophan, 40 to 60 grains (2.6–3.9 gm.) a day. The administration of salvarsan has been tried and favorable results have been reported. Bernheimer reports good results in one case from the injection of tuberculin. Intra-ocular injections of bichlorid of mercury, in the opinion of the author, should not be employed, although they have been highly endorsed. Subconjunctival injections have been recommended. The author's experience with them in this disease has not been encouraging. G. S. Derby and H. N. Pratt conclude, from their own experience and that of Zur Nedden, that the blood-serum of an individual with sympathetic ophthalmia may be of curative value when injected into another patient with the same disease. Not less than 1 ounce (30 c.c.) of the serum was used.

Under such treatment the affected eye will recover with useful sight, pass into atrophy or phthisis bulbi, or grow quiet, with the formation of complete annular adhesions of the iris to the capsule of the lens, which has become cataractous.

To improve vision under the last-named condition, iridectomy and iridotomy have been tried, but the results are usually unfavorable. Extraction of the cataractous lens, with iridectomy, also presents serious difficulties. For those cases in which a transformation of the iris, lens, and capsule into a tough, opaque, and inelastic tissue has occurred, Mr. George Critchett practised the following operation: The patient is placed under the influence of an anesthetic, a speculum is introduced, the globe is fixed, and a fine cutting needle is introduced through the cornea, its point being directed to the center of the capsule. This structure is penetrated by making a rapid rotary movement, on the principle of a gimlet. A second needle is introduced from the opposite side and the points separated from each other, the result being a rent in the center of the capsule and the escape of the soft lens matter. The operation must be repeated at proper intervals until a clear pupil has been obtained. It is suited to young eyes, although it may succeed in adults, as in one case in the author's practice. Care should be taken to avoid wounding the iris. With this operation the author has achieved gratifying success.

The **prognosis** of sympathetic ophthalmitis is essentially grave, although Alberti's statistics indicate that it is not as unfavorable as in former times, probably owing to modern antiseptic methods, and also that its manifestations are not so malignant. Useful vision may be retained, especially after sympathetic choroiditis (E. V. L. Brown). Complete and permanent recovery may sometimes occur; but the patient cannot be considered cured until at least a year has elapsed. Eyes in which papillitis is the manifestation of the sympathetic disease, and

which, according to Schirmer, never begins after removal of the exciting eye, are cured by enucleation of the originally injured eye, not immediately, like sympathetic irritation, but in the course of several weeks.

More frequently, especially in the forms which appear as an iridocyclitis or iridochoroiditis, the sight of the eye is lost and the organ shrinks. Excepting the cases of pure papillitis, those varieties which appear as a serous uveitis, and which retain this character of inflammation, afford the most satisfactory prognosis. It is extremely important to warn patients of the grave nature of this malady, and if an attempt is made to save an eye injured in the manner already described, it must be done with the full understanding of the serious risks which are undertaken, and the patient must be kept under constant observation.

CHAPTER XI

DISEASES OF THE CHOROID

Congenital Anomalies.—Two striking congenital anomalies occur in connection with the choroid:

1. *Coloboma of the choroid* is a large defect in the choroid, almost always in its lower part, and often associated with a similar vice of conformation in the iris.

Examined with the ophthalmoscope the deficient area appears as a glistening, pearl-colored patch, often irregular on its surface, owing to the development of several protrusions and corresponding intervening depressions, and bordered by an irregular pigment line. In some cases the retina may be recognized as a translucent veil covering the defect, and the retinal vessels occasionally pass into the depression; in others the retina is absent, and the defect will be represented in the visual field by a scotoma. The coloboma may include the optic-nerve entrance, either partially or completely, or may be separated from it by a bridge of healthy choroid. It may be confined to the area around the disk, or pass downward as far as it can be followed, and be connected with a similar defect in the iris, from which it is separated by a band of choroid tissue. Sometimes several defects are present in the same eye-ground. Imperfect closure of the fetal cleft (choroidal fissure) is the usual explanation of this condition. Some choroidal defects resembling coloboma apparently depend upon an intra-uterine choroiditis. According to E. T. Collins and W. Lang, all colobomas of the choroid may be explained by assuming that they are due to an abnormal adhesion of the retina to the mesoblast, which may take place either before or after the closure of the fetal cleft.

In addition to coloboma in the usual situations, similar defects have been described in the macular region (*macular coloboma*, see Fig. 160) and the nasal half of the eye-ground (B. A. Randall and the author), and for these defects, which do not involve the optic disk, Lindsay Johnson has proposed the name *extrapapillary coloboma*. *Atypical coloboma of the choroid* may also be situated upward and outward and probably in any part of the choroid. It is due to failure in the formation of blood-vessels in the inner part of the mesoblast surrounding the secondary optic vessel (Collins and Mayou). A striking developmental abnormality is one in which the entire choroid except a small area in the region of the macula is absent. To this condition the name *choroideremia* has been given. Both eyes are affected; the patients are night-blind (Nettleship).

2. *Albinism*, or a congenital want of pigment in the choroid and iris, is a deformity met with both in a *complete* and *incomplete* form. It depends upon a failure of pigment in mesoblastic tissue surrounding the

secondary optic vesicle and in the outer layers of the secondary optic vessel (Collins and Mayou).

The iris has a pink or pink and yellow appearance, due to the reflection of light from its own blood-vessels and from those of the choroid, which, in the most pronounced forms of the defect, can be seen with the ophthalmoscope down to their finer branches. The anomaly is most marked in early childhood, is almost invariably associated with lack of pigmentation in the hair, and is accompanied by nystagmus, amblyopia, and high grades of refractive defects. Usually these eyes are photophobic. In many instances albinism has been observed in several members of the same family, and seems to be hereditary. *Semi-albinism,*

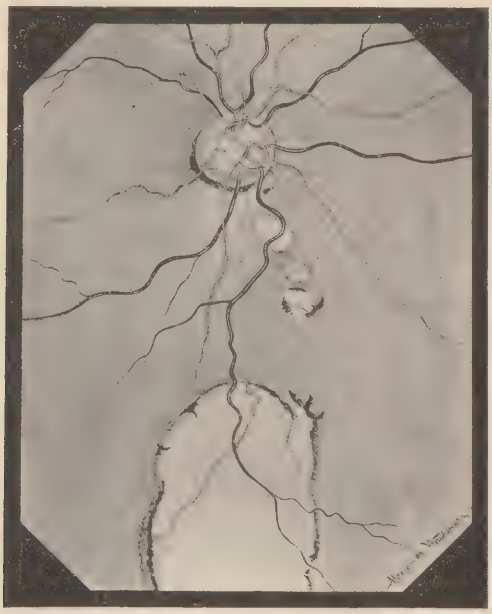


Fig. 152.—Congenital defects in the choroid; one large coloboma in the usual situation with two smaller areas between it and the disk.

especially in the periphery of the fundus, is sometimes observed in infants and may persist in adult life.

Hyperemia of the Choroid.—An actual hyperemia could be demonstrated only by finding a real distention of the vessels of the choroid, which usually are invisible, and the *congestion of the choroid,* described with myopic or asthenopic eyes, and as the result of exposure to bright light and heat, is more often a figure of speech than a proved pathologic condition.

In eyes subjected to prolonged strain, the result of uncorrected ametropia, certain changes in the normal appearance of the fundus arise which are usually described under the vague term "choroidal disturbance." We may, perhaps, assume hyperemia where the nerve-head presents distinct redness, which is imperfectly differentiated from the

unduly flannel-red appearance of the surrounding choroid, or where the choroid, instead of exhibiting its usual uniform red color, has changed into what has been denominated a "woolly choroid," with faint dark areas in the periphery, indicating the interspaces between the choroidal vessels, and more or less pronounced retinal striation surrounds the disk. This is a familiar picture in many cases of "eye-strain," and where the ophthalmoscope reveals, in addition to the other signs already described, an appearance as if fine pigmented grains had been scattered over the fundus, especially its central regions, the name "miliary choroidoretinitis" has been applied to it by Theobald. Similar lesions may follow exposure to great heat and light, and may be seen in the eyes of puddlers, etc.

Treatment.—In this condition, often associated with the subjective symptoms of aching eyes, some intolerance of artificial light and distinct asthenopia, the eye should be atropinized, dark glasses should be worn, and after the irritable condition of the fundus has sufficiently subsided a proper correction of the refractive error should be ordered. Internally, small doses of iodid and of bromid of potassium serve a useful purpose.

Choroiditis.—Under the general term *choroiditis* are included various types of inflammation of the choroid.

Causes.—Choroiditis, like iritis, may depend upon constitutional disorders, infections, toxins, and traumatisms, or upon disease in other portions of the eye. Choroiditis is often classified according to the probable etiology—for example, *syphilitic, tuberculous, traumatic,* etc., choroiditis. (Compare with pages 301, 302.)

Symptoms.—Certain symptoms, for the most part revealed only by the ophthalmoscope, are present:

1. Alteration in the uniform dull-red surface of the eye-ground caused by (*a*) the absorption of the pigment epithelium; (*b*) patches of pale-yellow color with ill-defined boundaries due to exudation (*recent choroiditis*); (*c*) patches of white color due to exposure of the underlying sclerotic (*atrophic choroiditis*); and (*d*) patches of black pigment, variously shaped, scattered over the fundus, and usually bounding the spots of atrophy (*pigment heaping*).

2. Absence of external manifestations indicative of the deep-seated disease, except where acute and purulent forms, in which the diseased process is not localized in the choroid, are accompanied by injection, chemosis of the conjunctiva, etc.

3. Changes in the transparent media (lens, vitreous) by the formation of opacities, as a secondary result of the choroidal disease.

Subjective symptoms peculiar to choroiditis do not exist.

Pain usually is not present except in purulent forms, and in such varieties as may be complicated with iritis.

Disturbance of vision is in direct relation to the situation of the lesions and the amount of atrophy. If the choroidal disease is peripheral, visual acuteness may be unaffected; if atrophic patches occupy the macular region, sight may be greatly diminished or practically obliterated. It is remarkable, however, that even in extensive diffuse choroiditis good vision may still be present. If the disease has caused second-

ary changes in the vitreous or lens, these add to the depreciation of visual acuteness.

Scotomas, both positive and negative, may be present. Contraction of the field of vision in certain types of choroiditis, and especially if secondary atrophy of the optic nerve has occurred. The displacement of the retinal elements overlying the diseased choroidal areas causes *metamorphopsia;* sometimes objects appear smaller than they really are, *micropsia;* sometimes larger, *macropsia.* In the early stages of choroiditis the patients are much annoyed by subjective symptoms of light—*i. e., photopsies.*

Diagnosis.—This is readily made by observing with the ophthalmoscope the appearances briefly summarized in paragraph 1 of the general symptoms.

Inasmuch as choroiditis, in the large majority of cases, is complicated with retinitis, it is difficult to decide whether the pigment lies in the choroid or retina. If the pigment mass is covered by a retinal vessel, and at the same time is situated in a deeper layer than this, its position is judged to be in the choroid; if the retinal vessel is covered by the pigment mass, and the latter is situated more anteriorly, its position is assumed to be in the inner surface of the retina, to which spot it has wandered through secondary involvement of the retina. Pigment characterized by a "lace-like pattern," or resembling bone-corpuscles, is always in the retina (Nettleship). A commingling of these positions in the same eye-ground is common.

Course, Complications, and Prognosis.—A choroiditis may be sudden in onset and pursue an acute course; for example, an acute choroiditis at the posterior pole of the eye resulting in a permanent myopia (see also page 129) or purulent forms of the disease.

More commonly the course of choroiditis is slow and chronic. Beginning with exudation or hemorrhage, it passes by slow stages through the period of absorption, atrophy, and pigment accumulation. It is by the last signs that a former choroiditis is recognized, and the changes are called "old choroiditis" or "choroidoretinitis."

The following structures are liable to become involved during the course of a choroiditis: The retina, which from its intimate association with the choroid through the pigment epithelium probably does not escape in any instance, and in many the association of disease is so close that we apply the term *choroidoretinitis* or *retinochoroiditis;* the optic nerve (*choroiditic atrophy*); the vitreous (*vitreous opacities*); the crystalline lens (*posterior polar cataract*); the iris (*iridochoroiditis*); and the sclerotic (*scleroticochoroiditis*).

The *prognosis* in choroiditis is always grave, and although careful treatment may preserve sight, in many instances great depreciation of vision and even blindness may ensue. Necessarily the prognosis as to vision depends upon the position of the disease and its relation to the macula.

Pathologic Anatomy.—In non-purulent forms of choroiditis collections of round cells are gathered in the choroid, especially along the ves-

sels between this membrane and the retina, and hemorrhagic extravasations may be seen. Organization of this round-cell exudation causes atrophy of corresponding portions of both choroid and retina, union of the two membranes, disappearance of the pigment layer of the retina except at the edges of the lesions, where it is proliferated, and wandering of the pigment cells into the retina along the lines of the vessels. In purulent choroiditis there is a dense cellular infiltration of the choroid, rapid involvement of retina and vitreous, panophthalmitis, and subsequent phthisis bulbi.

As already stated, choroiditis may be *acute* or *chronic,* and at one time was classified, according to the pathologic conditions, into plastic, serous, and purulent forms.

For the present purpose choroiditis may be divided into *superficial* and *deep* choroiditis, and a well-recognized classification may be adopted which places all forms under one of two heads: (1) *Non-suppurative exudative choroiditis* and (2) *suppurative choroiditis* and *iridochoroiditis.*

Treatment.—This, in general terms, demands perfect rest for the affected eye, protection from glaring light, and the administration of remedies indicated by the cause of the choroiditis. Further details will be reserved for the sections devoted to the several varieties of choroiditis.

Superficial Choroiditis (*Epithelial Choroiditis*).—Instead of the general dull-red appearance of the eye-ground, the larger vessels may be manifest as rather broad, reddish, or yellowish-red stripes, which traverse the fundus in an interlacing manner, and between which are the dark intervascular spaces, many of them having a lozenge-shaped appearance. This is due to the absorption of the pigment epithelium and the capillary layer which lies just beneath it.

In certain instances it is physiologic, and is commonly seen in the periphery of eye-grounds, often by preference occupying a space down and in from the disk.

It may be universal, the only portion of the eye-ground escaping being the region directly confined to the macula, and it then presents a striking picture to the ophthalmoscope. The larger vessels of the choroid-stroma pass in a sinuous manner across the eye-ground, bringing out into distinct relief the pigmented connective-tissue cells of the choroid proper which lie between them (consult Fig. 161, page 356). The atrophy is superficial and of itself does not disturb vision. Such appearances are seen in myopia; in "stretching eyes," when hyperopic refraction is diminishing or passing into myopic refraction; in glaucoma; and sometimes are associated with retinal disease—for example, pigmentary degeneration.

Deep Choroiditis.—1. **Diffuse Exudative Choroiditis.**—This occurs in an *acute* and in a *chronic* form—*i. e.*, the chronic form represents the ophthalmoscopic appearances commonly observed after subsidence of the acute process.

In the early stages of *acute choroiditis* the diseased areas are represented by yellowish-white, sometimes greenish-gray, exudations, which may be diffuse, circumscribed, or disseminated, and which shade gradu-

ally into the surrounding eye-ground, or which may be fringed with pigment, small eroded areas, and hemorrhages.

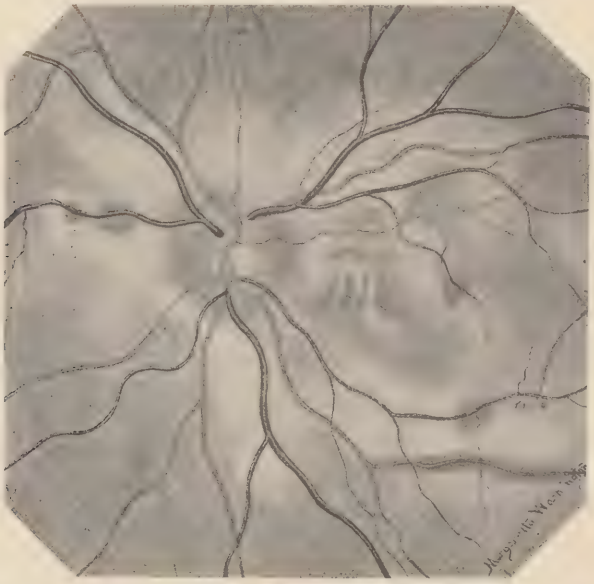


Fig. 153.—Acute choroiditis with wide-spread, fog-like exudations.

Later these areas of exudation undergo absorption and metamorphosis, and some of the following conditions are present, which may be named *chronic choroiditis:* Instead of the normal red of the eye-ground the ophthalmoscope reveals white or yellowish-white plaques, sometimes separated by partly normal choroid, more often running into one another until a huge expanse of exposed sclera is visible throughout the fundus.

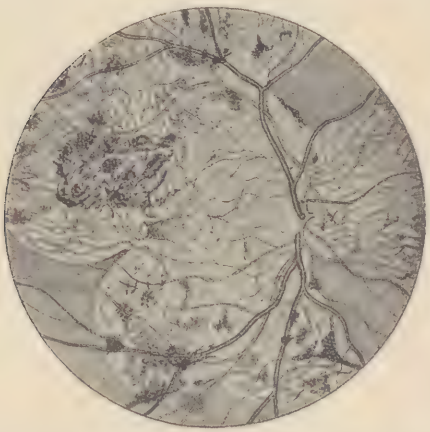


Fig. 154.—Diffuse exudative choroiditis with choroidoretinitis (E. von Jaeger).

The white patches appear speckled because numerous pigment masses of black color are collected upon them, irregular in form, sometimes gathered in lumps, sometimes assuming variously shaped groups. They lie beneath the retinal vessels for the most part, although usually pigment will be found collected upon these retinal vessels showing the participation of the retina in the process (*choroidoretinitis*, Fig. 154).

In other patches the atrophy has not been sufficient to expose the glistening white sclera, and there will be found the lesions of superficial choroiditis, namely, band-like, orange-yellow, or light red vessels, freely anastomosing with each other, and, between them, the pigmented epithelium. In still other spots yellowish exudations are evident, which represent the earlier stages of the process already described. In these circumstances all the stages from yellowish extravasation to complete atrophy are visible.

2. **Disseminated Choroiditis.**—Another form which may be looked upon, according to a classification adopted by some authors, as the circumscribed variety of the type just described, is that which is known as *disseminated choroiditis*.

In this type, usually beginning in the periphery, but gradually approaching the center of the eye-ground, numerous round or oval spots surrounded by black margins are found. The white center of the spot is the exposed sclera; the black margin, the altered pigment. Instead of a white center there may be a single black mass, in its turn encircled by a yellowish ring. Where the spots assume a punched-out look, as if a sharp instrument had cut out the tissue down to the sclera, the margins of the incision being bordered with pigment, the appearances are characteristic.

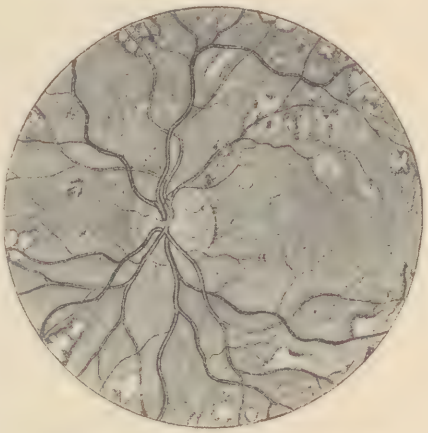


Fig. 155.—Disseminated choroiditis (de Wecker).

These spots of disseminated choroiditis vary greatly in number. There may be only one or two, or the eye-ground may be dotted over with them. Between the spots the choroidal tissue is comparatively healthy. The earlier stages of such spots consist in small, yellowish or greenish-gray exudations, which gradually absorb, leaving the atrophic marks which have just been described (Fig. 155).

Vitreous opacities are often present, either faint and floating, or large, string-like, and membranous. There may also be cataract at the posterior pole of the lens.

The optic nerve may become affected in the later stages of deep choroiditis and undergo a species of atrophy to which the name *choroiditic atrophy* has been applied. The edges of the disk are slightly hazy, the color a reddish yellow, and there is contraction of the retinal vessels. Disseminated and other forms of choroiditis are often associated with *secondary pigmentation of the retina*, and the pigment patches not uncommonly resemble those seen in pigmentary degeneration of the retina.

These pigmentations are especially noteworthy in forms of disseminated choroiditis due to syphilis.

3. **Circumscribed Plastic Choroiditis** (*Localized Exudative Choroiditis; Choroiditis with Descemetitis*).—Areas of choroiditis are not infrequently encountered in young persons and young adults—that is, from fifteen to thirty years of age—which ophthalmoscopically do not differ materially from those types already described, except in their circumscribed character. Usually there is a large, bluish-white patch of effusion, generally denser in its center and thinning off at its margin into the healthy fundus. The diseased area may be close to the disk (Hill Griffith), near or at the macula, or in the periphery (Friedenwald). The early exudative stage is followed by erosion and atrophy, but the course is compara-

Fig. 156.—Disseminated choroiditis and optic neuritis with retinal hemorrhages in a syphilitic patient in the Orthopedic Hospital. The choroiditis is of long standing, the neuroretinal lesions a fresh implantation.

tively benign, and, unless the macular region is involved, there is no distinct depreciation of central vision. Not uncommonly keratitis punctata (descemetitis) and vitreous opacities accompany the condition (see also page 325). Not infrequently rise of intra-ocular tension may be present and an attack of *acute glaucoma* may be precipitated, especially if a mydriatic is employed.

Plastic choroiditis near the disk may give rise to an appearance similar to that of optic neuritis. One variety described by Jansen has been named by him *retinochoroiditis juxta papillaris;* its position is interpreted in the visual field by a scotoma which joins the blind-spot.

4. **Anterior Choroiditis.**—To this condition reference has been made in the description of parenchymatous keratitis (see page 269). The lesions are situated far in the periphery, and may exist as a special form of disease, or result from an extension backward of affections of the iris and ciliary body.

Causes.—The cause of deep choroiditis, either diffuse or disseminated, is acquired syphilis in a great number of cases, and the disease appears from six months to two years after the initial infection. Sometimes it is postponed to a much later period (tertiary period). Opacities in the vitreous are common in syphilitic choroiditis. Although certain choroidal lesions have been looked upon as especially characteristic of syphilis, it is not safe to attempt to make a diagnosis of syphilis simply by the appearances of any of the varieties of choroiditis. Diffuse syphilitic choroiditis depends upon a filtration of the toxin of syphilis, or perhaps a dissemination of its active agent (Spirochæta pallida) throughout the tissue of the choroid. If the deposit of the toxin remains localized the circumscribed varieties of the affection arise. Disseminated choroiditis, choroidoretinitis, and secondary pigment degeneration of the retina are seen in children the subjects of hereditary syphilis. Choroiditis due to acquired syphilis usually affects both eyes. The Wassermann test should be applied to all cases of choroiditis.

A disseminated choroiditis (*hereditary choroiditis*) affecting both eyes is occasionally encountered as a family disease independently of syphilis and associated with disorders of the central nervous system (Hutchinson), and *familial chorioretinitis* has been observed. Patches of choroiditis are found in the eyes of children born with cataract.

A choroiditis quite indistinguishable from the forms described may result from an injury (*traumatic choroiditis*). As the result of concussion injuries of the globe *pigmented choroiditis* may arise in the form of scattered areas of variously shaped pigment masses, usually of small size, sometimes granular, interspersed with small erosions and spots of atrophy.

Many cases of choroiditis, especially of the disseminated variety, as well as of the diffuse and localized exudative manifestations, are due to tuberculosis (*tuberculous choroiditis*). This etiologic factor can be demonstrated by tuberculin injections, which are followed by local reaction.

Choroiditis has also been ascribed to disturbances of nutrition, metabolic disorders, nephritis (*albuminuric choroiditis*), diseases of the liver (*ophthalmia hepatica*), anemia, chlorosis, acute infectious diseases, and to infections arising from the nasopharynx, accessory sinuses, and the teeth (pyorrhœa alveolaris). In acute plastic choroiditis the following etiologic factors have been reported: acute tonsillitis, oral sepsis, intestinal toxemia, auto-intoxication, typhoid fever and other infectious fevers, and pneumonia. Gradle suggests that the infecting material from the accessory sinus or other focus of infection may gain access to this region through the posterior ciliary vessels. (For additional causes, see page 324.)

The **prognosis** is always grave if the process is an extended one and the macula involved; it is best in the syphilitic cases and in some forms of acute plastic choroiditis, in which the results of treatment are most satisfactory.

Treatment.—This depends upon the cause. If it is syphilis, inunctions of mercurial ointment should be prescribed, to be followed by iodid of potassium, or the mercury may be given by the mouth in the form of the protiodid, or by the hypodermic method. Later, a prolonged course of bichlorid of mercury combined with tincture of iron is advisable. *Salvarsan* and *neosalvarsan* in the later states of choroiditis have not seemed to the author to be of much value; in acute forms their action is more satisfactory (compare page 310). Subconjunctival injections have been recommended. They may be composed of bichlorid of mercury (1 : 2000–4000), cyanid of mercury (1 : 5000), or physiologic salt solution. Pilocarpin sweats may be tried, and in non-syphilitic cases their effect is sometimes strikingly favorable; in old cases strychnin and the galvanic current have been advised. If tuberculosis is the suspected or definitely established cause, *tuberculin* should be administered (see page 318). Certain cases of intra-ocular tuberculosis (*choroiditis*), especially characterized by chronicity of the lesions, are distinctly amenable to this treatment. All close work must be forbidden; the eyes should be protected with dark glasses. Should a mydriatic be employed in the treatment of acute choroiditis, its effect on the intra-ocular tension must be carefully watched and frequent tonometric tests are advisable. Dionin is of service; also subconjunctival injections. Naturally, general medication should be governed by the probable etiologic factors, and what has been written on page 328 applies to the disease now under discussion, and in acute choroiditis, especially of the circumscribed variety, great care should be exercised in a search for focal infection, particularly in the buccal mucous membrane.

Central choroiditis is the name applied to choroiditis confined to the region of the macula; its manifestations are numerous.

There may be an irregular patch of exudation, semi- or completely atrophic, and bounded by pigment. This is recognized objectively by the ophthalmoscope, and subjectively by a scotoma in the field of vision. Occasionally the area consists of an epithelial atrophy, either with a well-marked border, somewhat irregular in outline, or with a border less sharply marked, and with pigment distributed over the surface of the defect. Sometimes the lesions consist of areas of yellowish exudation, interspersed with small round and linear pigment masses and dot-like hemorrhages. Kipp calls especial attention to *hemorrhagic central retinochoroiditis* in non-myopic eyes, characterized by an oval or round area, surrounded by hemorrhage.

Again, the macula may be occupied or surrounded by a large white patch, the rest of the eye-ground being normal. Occasionally the area is entirely circular and the deep vessels exposed, or they may be atrophied and converted into white lines (*sclerosis of the choroidal vessels*). Pigment is usually absent. To these types of choroidal change the same *senile*

areolar atrophy of the choroid is usually applied. Extensive *sclerosis of the choroidal vessels*, which are apparently converted into white lines, with some pigmentation in the periphery of the eye-ground, but with normal disks and retinal vessels, has been reported as a *family disease*, that is, it may occur in several members of the same family. A form of choroiditis especially described by Förster is known as *choroiditis areolata*, and affects the region of the disk and the macula. The spots are numerous, but larger than those in disseminated choroiditis. Their centers may be white, and are usually black rimmed and sometimes undermined. Occasionally the macula is covered with a greenish or grayish plastic exudate, of various shapes, often surrounded by a rim of hemorrhage and later by a zone of erosion or atrophy—probably a late stage of plastic choroiditis (see page 350).

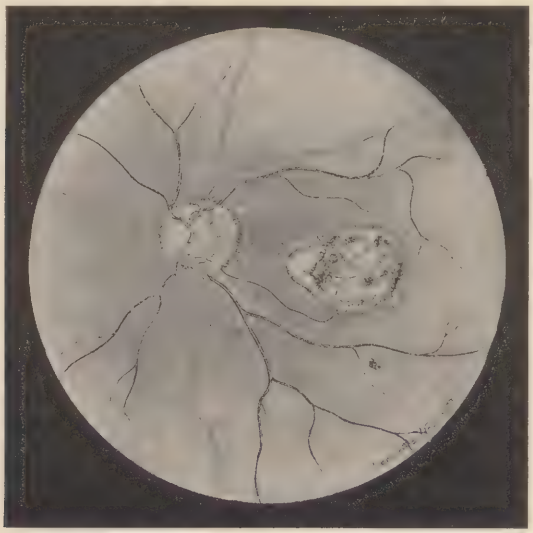


Fig. 157.—Central atrophic choroiditis; on the temporal side of the disk there is a semi-atrophic area—the so-called conus (from a patient in the Philadelphia General Hospital).

In the same region there is observed another variety of the disease, first described by Tay and Hutchinson as central *senile guttate choroiditis*, marked by the appearance of numerous white, glistening dots, somewhat resembling the earlier stages of albuminuric retinitis (Nettleship) and always symmetric, though sometimes an interval of time elapses before the implication of the second eye. The white spots are due to colloid degeneration and calcareous formations in the choroid, and are associated with secondary involvement of the retina. Occasionally the macular region contains an oval or circular patch of dense grayish-white or yellowish-white tissue, which lies beneath the retina and seems to be in the choroid, and which, according to Nettleship, belongs to this group of central senile choroidoretinitis. Usually there are contraction of the field of vision and negative scotoma. Large areas of *colloid change* may

also occur without disturbance of vision (*verrucosities of the choroid*), Fig. 158.

It is important, if possible, to recognize all forms of central choroiditis before a cataract operation is performed. They may be suspected if there is imperfect central fixation for light, but really can be positively determined only while the cataract is still incipient and the ophthalmoscopic examination is possible.

Causes.—Central choroiditis of inflammatory type may be caused by syphilis and also by blows upon the eye. Chronic atrophic choroiditis in this region is seen in myopia, and Gould has described macular choroiditis as the result of uncorrected ametropia and insufficiency of the internal recti muscles, even in non-stretching eyes (*ametropic choroiditis*). Nettleship believes that central senile choroidoretinitis, in its various manifestations, depends chiefly upon disease of the posterior ciliary arteries, either the trunks themselves or the branches which perforate

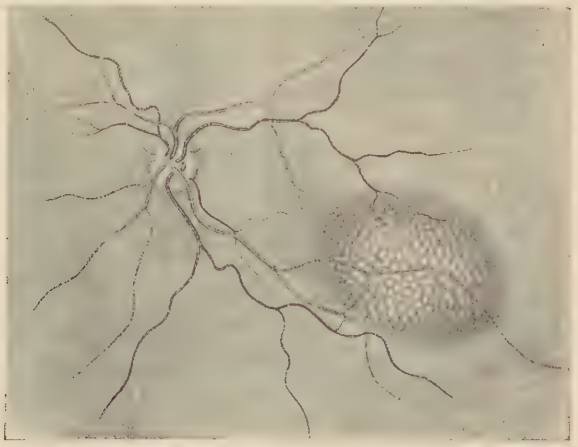


Fig. 158.—Colloid change in the macular region.

the sclerotic near the optic nerve, and that any affection of the retina itself is secondary.

Treatment.—In the syphilitic variety the usual remedies are indicated. In types connected with refractive error the best possible correction should be given and absolute eye-rest enjoined. In the senile varieties, both the ordinary and the guttate types, treatment appears to have no influence.

Unclassified Forms of Choroiditis.—Besides the diseases of the choroid which have been described others appear which cannot be definitely classified:

Large patches of atrophy not located in special portions of the choroid, resulting probably from the absorption of former hemorrhages, or, perhaps, tuberculous areas; *hemorrhagic choroiditis* occurring, as pointed out by Hutchinson, especially in young men, and resulting in numerous spots of atrophy which are not readily distinguished from those

of the syphilitic variety; yellowish or other spots of choroidal disease, which have been attributed to the action of intense light or the glare

Fig. 159.—Central cone-shaped lesion in choroid, surrounded by lines of edema (patient in the University Hospital).

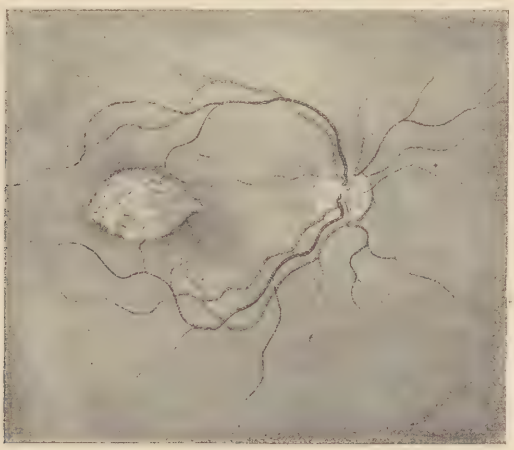


Fig. 160.—Macular coloboma (compare with Fig. 157).

of heat; slight macular changes in the form of small yellowish or maroon-colored spots, sometimes with a few scattered pigment granules in the immediate vicinity of the fovea, which do not affect vision and are

unnoted by the patient. These have been attributed by some authors to the influence of abnormal refraction, but are sometimes seen in association with transient albuminuria, and probably represent small spots of degeneration due to vascular disease, perhaps of the short posterior ciliary arteries which supply the region of the macula.

Myopic Choroiditis.—Atrophy of the choroid, commonly of a local character, occurs in severe or, as it has sometimes been called, malignant myopia, and is observed either in connection with or surrounding the nerve-head. It is caused by the elongation which occurs at the posterior pole of the eye, and receives the name *posterior staphyloma;* if the disk is entirely surrounded by the area of atrophy the name *annular posterior staphyloma* or *circumpapillary atrophy* is suitable (Fig. 161).

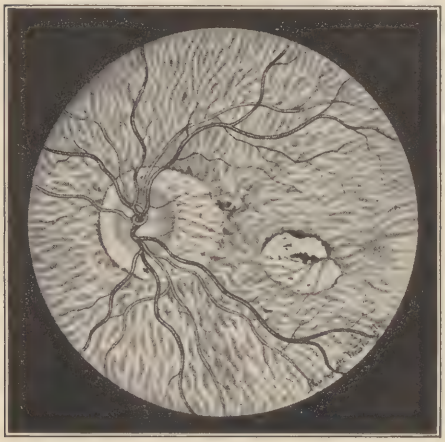


Fig. 161.—Myopic choroiditis. The cut illustrates posterior staphyloma—the white area surrounding the nerve: atrophic choroiditis in the macula—the white patch bordered by pigment in the central part; and general exposure of the choroidal vessels by absorption of the retinal pigment epithelium.

The term *scleroticochoroiditis posterior* is also applied to this variety of choroidal change, just as *anterior scleroticochoroiditis* is the name given to the inflammatory affection which attacks circumscribed portions of the anterior part of the choroid, with corresponding portions of the sclerotic, and which, in aggravated instances, may give rise to staphylomatous bulging and gradual loss of vision by opacity of the vitreous and cornea (see page 291). Hirschberg objects to the term "myopic choroiditis," as he believes the lesions are mechanical and not inflammatory in origin.

Semi-atrophic and *atrophic crescents* (often inaccurately called "conus") also appear at the outer margin of the disk in astigmatic eyes, and in eyes undergoing change owing to the distention of their coats from too close work aggravated by imperfectly or improperly corrected errors of refraction. In hyperopic and emmetropic eyes narrow white crescents, usually at the temporal side of the disk, are often evident. These are the so-called *scleral crescents.*

In the macular region in myopia there may be very decided semi-atrophic or atrophic patches having the general characteristics of the spots already described, and greatly interfering with vision. The process begins in the form of small rents which gradually coalesce into an atrophic patch. In like manner this area may be involved by a hemorrhage in progressive myopia, which after absorption leaves impaired vision, owing to the damage of the overlying retina. The vessels of the

choroid are exposed by maceration and absorption of the retinal pigment epithelium, causing the appearance described under superficial choroiditis (see Fig. 161, and also page 347.)

Suppurative Choroiditis and Iridochoroiditis.—Acute iritis occasionally becomes complicated with inflammation of the choroid (see page 305), and a chronic type of iridochoroiditis, which tends to loss of vision and shrinking of the eyeball, has been described (see pages 315 and 326).

The present disease, however, is distinguished by a suppurative process between the retina and choroid, which extends into the vitreous and spreads into the entire uveal tract.

Symptoms.—There are edema of the lids, chemosis of the conjunctiva, haziness of the cornea, inflammation of the iris and ciliary body, and, it may be, hypopyon. If there is sufficient transparency of the media, a mass of exudation may be seen behind the lens in the vitreous, giving rise to a yellowish reflection when viewed by transmitted light (*pseudoglioma, destructive ophthalmitis;* see also page 414). At first the tension may be raised and the anterior chamber is shallow; later the tension is lowered.

In addition to these objective symptoms there are severe brow-pain, tenderness of the globe, loss of vision, and constitutional symptoms, as chill and fever.

The ultimate result depends upon whether the disease remains localized in the uveal tract and vitreous, or spreads to all the tissues of the eyeball. In the former case the inflammatory symptoms subside, the pain lessens, the intra-ocular tension is lowered, and the eyeball gradually shrinks.

In the latter case the inflammation spreads, the edema of the lids and chemosis of the conjunctiva are intense, the pain severe, and the constitutional symptoms—fever, chills, nausea, and vomiting—are very marked. The inflammation involves Tenon's capsule and causes protrusion of the globe, which is pressed against the swollen lids until these can scarcely be separated on account of the swelling and edema. Finally, rupture of the sclera or sloughing of the cornea occurs, the purulent matter finds a vent, the pain subsides, and in about six weeks the ball is soft, sightless, shrunken, and free from pain. The second outcome of purulent choroiditis is known as *panophthalmitis*, and the ultimate result is *phthisis bulbi.*

Causes.—*Suppurative choroiditis*, or *iridochoroiditis*, may be caused by the introduction of pathogenic microbes in the same manner as in purulent cyclitis—that is, the infection comes from the outside. It is, in short, an exogenous infection. In these circumstances it may arise as the result of perforating wounds and injuries; operative wounds which have become infected—for example, cataract extraction, sloughing ulcers and abscesses of the cornea, and prolapse of the iris and thinned cystoid corneal cicatrices, those, for example, which are created by corneoscleral trephining, a diminutive aperture having formed, permitting the entrance of micro-organisms.

Suppurative choroiditis may also be caused by embolism from a microbic area, and produces the condition which is known as *metastatic ophthalmitis*. From the etiologic standpoint, following Axenfeld's classification, metastatic ophthalmitis may result from puerperal pyemia, which is its most frequent cause; from surgical pyemia, which includes all cases which arise from injury, operations, and local purulent areas, even where the last-named conditions are non-traumatic, but have an internal origin, and have their situation in the mucous membrane of the digestive, pulmonary, and urinary organs (Groenouw); from cryptogenetic septicopyemia, that is, the point of entrance of the infection has not definitely been determined, and, finally, from infectious diseases, particularly pneumonia, influenza, measles, scarlet fever, diphtheria, and small-pox. The disease may also result from cerebrospinal meningitis, basic meningitis, dysentery, bronchitis, whooping-cough, inflammation of the umbilical vein, and thrombosis of the orbital veins. It may be bilateral or unilateral, and the puerperal cases usually develop during the first two weeks of the disease, but may be detained until the seventh week. Ulcerative endocarditis is a frequent complicating factor.

Pathology.—Examination of eyes in which suppurative choroiditis has occurred shows the presence of a thick purulent infiltration of the choroid, involvement of the overlying retina, and sometimes conversion of the entire vitreous into a purulent material. In the metastatic variety of the affection the septic masses enter into the capillaries of the eye. Sometimes the retina is exclusively or, at least, first affected, later the uveal tract is also involved.

Fuchs' investigations of the anatomic changes in inflammation of the choroid and those which result from infection of the vitreous indicate that the inflammation spreads to the inner lining of the vitreous, especially to the *pars ciliaris retinæ* and to the retina itself. Purulent retinitis results, and the choroid is seriously involved where the inflamed retina remains in contact with it. To this process he gives the name *endophthalmitis septica*.

Streptococci, staphylococci, and sometimes Fränkel-Weichselbaum pneumococci have been found, and in many cases of panophthalmitis not necessarily metastatic in origin special bacilli are present, some of which have been determined to have pathogenic significance. This is particularly true after injuries of the eye, and in a number of instances those organisms have been found to which Haab has given the name *panophthalmitis bacilli*, and which belong to the group of the "hay bacilli."

Prognosis.—This is most unfavorable, and almost invariably a shrunken eyeball is the result of the inflammation. A few cases of recovery from suppurative iridochoroiditis following cerebrospinal meningitis have been recorded. The termination of destructive ophthalmitis in children is usually not fatal, but a few deaths have occurred, generally from meningitis (see also page 414). In bilateral cases of puerperal metastatic ophthalmitis the mortality is exceedingly high, only a few authentic cases of recovery being on record.

Treatment.—In the early stages antiphlogistic treatment may be of service in robust cases—blood-letting from the temple, a sedative fever mixture, with sufficient morphin to relieve pain, and locally, frequently changed ice compresses. In later stages hot fomentations are better, a square of lint soaked in heated bichlorid solution being applied to the eye; and internally, opium and quinin in full doses are indicated. If there is much pain before spontaneous rupture has occurred, a free incision into the sclerotic will bring relief. The methods of treatment to prevent the spread of septic processes after injury have been described on page 296.

Surgeons differ in regard to the advisability of enucleating the globe during the acute stages of panophthalmitis, some operators declining to perform excision in such circumstances, in the belief that meningitis is liable to follow, while others do not recognize this danger.

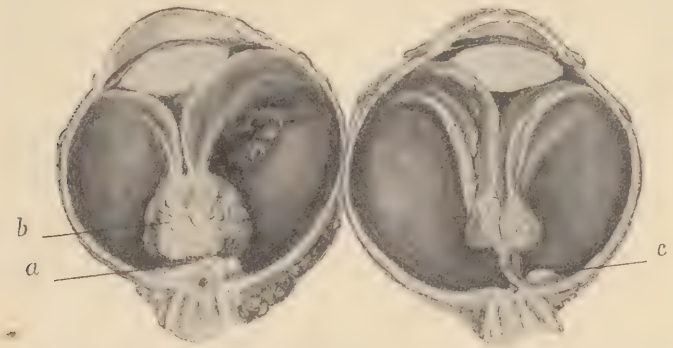


Fig. 162.—Leukosarcoma of choroid, showing at (*a*) constriction which marks where choroidal capsule was ruptured and where retina became adherent, being pushed forward with growth of upper part of tumor (*b*), which assumes a mushroom shape. At (*c*) choroidal origin of growth is seen.

The author does not hesitate to enucleate an eyeball in which there is suppuration if the surrounding orbital tissues are not yet involved in the process; but agrees with Pooley that where the process has reached a great height, where there is purulent infiltration of the orbital tissues, and where the affection has begun posteriorly, as in some varieties of septic iridochoroiditis, the operation of enucleation is surrounded by dangers. In a certain number of cases it has been followed by meningitis. Therefore under these conditions evisceration is preferred. But even after evisceration there may be a great accumulation of inflammatory products behind the scleral cup, and to these a vent must be given.

Tumors of the Choroid.—The most frequent neoplasm of the uveal tract and, for the present purposes of description, of the choroid is *sarcoma*. Most commonly it appears as a pigmented growth (*melanosarcoma*); more rarely (1 in 8, according to W. C. Rockliffe) without pigment (*leukosarcoma*).

Sarcoma of the choroid, according to E. Pawel, is most frequent between the ages of fifty and sixty. A good many cases, however, occur

at an earlier period than this, but the disease is rare under the twentieth year. Men are more frequently affected than women, and the left eye, according to some statistics, is more apt to be involved than its fellow.

Pathology.—The growth usually is circumscribed, and has a spheroid form as long as the choroidal capsule remains unbroken. Sometimes it assumes a cake-like shape, and occasionally the form of a mushroom. Rarely, there is diffuse sarcomatous infiltration of the choroid.

Sarcoma of the choroid is almost invariably a primary growth; but the choroidal coat may be,

Fig. 163.—Macroscopic appearance of a pigmented choroidal sarcoma—flattened growth or so-called cake-like form. One extrascleral nodule.

Fig. 164.—1, Pigmented flat sarcoma; 2, cyst of retina; 3, detachment of retina in portion of eye opposite to position of tumor.

though very rarely, affected by a metastasis occurring from a tumor in some other portion of the body—for example, the mediastinum (A. V. Meigs and the author).

The tumor develops from the outer layers of the choroid, and grows inward, detaching the retina. The cells are round or spindle formed, or occasionally of a large endothelioid type, provided they develop from the endothelial linings of the lymph-spaces. They are usually pigmented, the density of the pigmentation depending upon the participation of the choroidal stroma cells in the proliferative process. Usually there are many broad vessels around which the cells may be grouped. Intraocular sarcomas may undergo necrosis owing to the death of groups of cells at a distance from blood-vessels from insufficient oxygenation. Toxins are liberated which devitalize the surrounding cells (Fuchs). In the second stage secondary glaucoma occurs, and occasionally plastic iridocyclitis appears and results in atrophy of the globe. A tumor may grow in a phthisical eye, and, as Leber has pointed out, an eye which is the seat of a growth may become phthisical and the tumor itself cease to grow for a time. Coppez divides the primary new growths of the choroid into—(1) *Interfascicular endotheliomas* which develop from the

endothelial cells of the lymph-spaces; (2) *peritheliomas* (angiosarcomas) which arise from the perithelial cells of the blood-vessels; (3) *sarcomas* of various characters which grow from the proper cells of the choroid and the adventitia of the blood-vessels. *Alveolar sarcomas*, also called endotheliomas or intravascular angiosarcomas, are rare as compared with perivascular sarcomas of the choroid. It is probable that in these alveolar types the greater mass of the tumor is formed by the proliferation of endothelial cells.

Diffuse sarcomas of the choroid are classified by Parsons into two subgroups—*flat sarcomas* and *ring sarcomas*. They are characterized by an infiltrating tendency, as opposed to the formation of a definite tumor. They exhibit large round or polygonal cells, alveolar or plexiform arrangement, hyaline and myxomatous degeneration, and extension along the perivascular lymph-spaces. According to Parsons, they should be

Fig. 165.—Pigmented sarcoma at *a*; retina detached and folded.

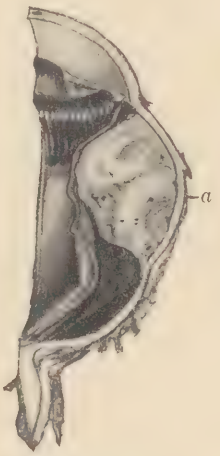


Fig. 166.—Leukosarcoma at *a*; retina detached.

considered as endotheliomas, and they spring from the lining cells and proliferate in the spaces which they line. The onset of glaucoma is early in diffuse sarcoma.

Parsons has also called attention to certain *anomalous sarcomas of the choroid* which bear a microscopic similarity to organizing blood-clot and are of comparatively low malignancy. According to him, if they are not excised, they shrink, and represent the tumors before referred to which have been found in phthisical eyes. Destructive hemorrhage may occur in choroidal sarcomas, as has been specially pointed out by Verhoeff. Hemorrhages of this character may be responsible for the sudden attacks of glaucoma which are often seen in choroidal sarcoma. Occasionally sarcomas of the choroid may take their origin in *melanomas*, just as they do in the iris and ciliary body. A good example of such an origin has been reported by the author and E. A. Shumway. The smallest sar-

comas on record have been described by Fuchs (in one instance the growth being between 0.7 and 0.8 mm. in the horizontal and vertical diameters), and Shumway and the author have also observed a very small sarcoma of the choroid (4.4 mm. in the anteroposterior diameter and 0.9 mm. in thickness).

Symptoms.—The life history of a sarcoma of the choroid has been divided by systematic writers into four periods: The first, the quiet period; the second, the inflammatory period; the third, the extra-ocular period, or that stage when the growth bursts through the scleral boundary; and fourth, the period of metastasis.

In the *first stage* the disease resembles a detachment of the retina, this membrane being pushed forward by the underlying elevation, the whole being surrounded by a serous effusion. Beneath this retinal covering the brownish mass may sometimes be recognized, covered by irregular choroidal vessels, except in the non-pigmented varieties. If the growth is situated far forward, it is sometimes possible to examine it by means of oblique illumination through a dilated pupil. There is a corresponding defect in the field of vision, and the sight of the affected eye is diminished in accordance with the situation of the tumor. Should this be peripheral, the central vision at this stage may not be seriously affected. The first stage usually lasts from six to twelve months, but rarely may be prolonged to five years. Occasionally sarcoma of the choroid produces symptoms resembling *tenonitis* (Kipp and the author).

In the next period of the history of this growth, or the *inflammatory* or *glaucomatous stage*, symptoms of increased tension which depend upon alterations in the angle of the anterior chamber arise: pain in the brow, anesthesia of the cornea, shallowing of the anterior chamber, and dilatation and tortuosity of the perforating episcleral vessels. Ophthalmoscopic examination is no longer possible, the localized detachment of the retina becomes general by increased serous effusion, the lens may become cataractous, and a severe iridocyclitis may supervene.

As the growth continues, the sclera is ruptured and the surrounding tissues are involved (*fungus state* or *stage of episcleral tumors*). It may pass backward into the brain, or secondarily affect the optic nerve, but more commonly the last, or *metastatic stage* (*stage of generalization*), develops; distant organs are attacked by growths of similar histologic character, the liver far more frequently than other organs, but also the spleen, intestines, and even the lungs. Metastasis to the liver need not necessarily be delayed until the tumor has burst, at least visibly, through the scleral boundary. The most extensive secondary sarcoma of the liver which has come under the writer's notice developed from a small sarcoma of the choroid, in which there was no external manifestation, but in which a few fragments in the orbital tissues appeared to be of suspicious nature after the removal of the eye.

Diagnosis.—It is necessary to differentiate sarcoma of the choroid from glioma of the retina. To this reference will be made in a future section.

In the early stages choroidal sarcoma may be mistaken for idiopathic detachment of the retina and detachment of the choroid. In retinal detachment there is usually a history of sudden onset, and the ophthalmoscope may reveal undulations of the folds of the detached retina with the movements of the eye, vitreous opacities, and signs of choroiditis. Moreover, the field is frequently less sharply defective than in choroidal sarcoma. Parsons, impressed with the fact that early detachment of the retina occurs in many cases of sarcoma of the choroid, urges in all instances of apparently simple detachment of the retina that a most careful search for tumor should be made by the methods already described, and especially by *transillumination of the sclerotic (diaphanoscopy)*, by means of which the most satisfactory results are obtained. Various instruments have been designed, notably those of Leber, Sachs, and Würdemann. The eye having been cocainized, the point of the instrument is passed over all areas of the exposed sclera. In the absence of a growth the red glare in the pupil remains undisturbed and bright; if a growth exists, the passage of the light is obstructed as the point of the instrument is placed over the region beneath which it is situated, and the pupil remains dark. In order to detect tumors situated far posteriorly, Lancaster has mounted a small Tungsten lamp at the end of a flexible copper tube attached to a posterior flashlight battery. Through an incision in the conjunctiva and capsule of Tenon the lamp can be carried behind the eyeball and its posterior segment thus transilluminated. The value of *ophthalmodiaphanoscopy* in these circumstances has been described on page 110.

Choroidal detachment is rare, the history is different from that of sarcoma, and the characteristic vessels of the choroid can usually be recognized beneath the vessels of the retina.

Too much reliance cannot be placed upon the tension of the eyeball as a distinguishing sign between sarcoma and retinal detachment, because intra-ocular tension may be unaltered in each instance, although, as C. Devereux Marshall has shown, it is probably never diminished (as it often is in retinal detachment) in undoubted cases of choroidal sarcoma, while it may be reduced in cases of sarcoma of the ciliary body.

In the stage of increased pressure the disease is to be distinguished from glaucoma by observing the suddenness of the onset of inflammatory symptoms—in the latter disease without antecedent history of poor vision—and the fact that in the glaucomatous eye there are remissions in the acute symptoms, and that the tension is somewhat amenable to the miotics.

Prognosis.—Removal of an eye for choroidal sarcoma results in a cure in from 25 to 30 per cent. of the cases, although statistics on this point vary greatly. Hirschberg's published statistics may be briefly summarized as follows: Local recurrence, 2.5 per cent.; metastasis, 41.5 per cent.; cure, 56 per cent. He points out that statistics show a steady improvement in so far as permanent recovery after enucleation for sarcoma of the choroid is concerned. His own earlier operations yielded

only 35 per cent. of recoveries. Metastasis to internal organs is the most usual cause of death and generally takes place within two years after operation. The stage at which enucleation is performed does not certainly influence the occurrence of metastasis, although, as Hirschberg maintains, operation at the very earliest stage should be urged. It is usually stated that very vascular and round-celled sarcomas are more fatal than other varieties. Prognosis is better in young than in aged subjects. Local recurrence is much less frequent than metastasis; it is prevented by prompt removal of the eye. If there is no recurrence or metastasis within four years after enucleation of the eye, this complication becomes unlikely, although exceptions to this rule have occurred, and metastasis has been noted even after seven years.

Treatment.—From what has been said it is evident that the only treatment is prompt enucleation. The optic nerve should be severed as far back in the orbit as is possible. It may be necessary to remove the entire contents of the orbit. The treatment of choroidal sarcoma by radium has not yielded satisfactory results.

Rare forms of tumor of the choroid are the following: *Cavernous angioma*, *telangiectatic sarcoma*, *adenoma*, and *enchondroma*. Angiomas of the choroid (about 21 are on record) may occur in connection with nevi of the face.

Carcinoma of the Choroid.—The tumor (a comparatively rare growth, about one-third being bilateral) is of rapid development and generally appears as a flat growth in the neighborhood of the macula. In the majority of instances it represents a metastasis from a carcinoma of the mammary gland (39 times in 64 cases collected by Suker and Grosvener); the primary neoplasm has also been situated in the lungs, pleura, stomach, liver, thyroid, mediastinal glands, suprarenal gland, prostate, and ovary.

Tubercle of the Choroid.—Tubercles appear in the choroid as yellowish-white spots, varying in size from 1 to 1.5 mm., occasionally larger, and usually, though not necessarily, associated with similar growths in the meninges (tuberculous meningitis). Repeated examination is required for their detection, and even then they may escape observation, owing to their diminutive size (*choroidal dust*). The facility of their detection has been much enhanced since the introduction of electric ophthalmoscopes. They have been frequently found in post-mortem examinations.

Tubercles, known as miliary tubercles, are distinguished chiefly by their color, which has been described as of a dull yellowish white in the center, encircled by an ill-defined rose-colored area (Horner). Usually there are no pigmentary changes in the immediate neighborhood, but pigment bodies may surround the nodules if they are prominent (Bach). They are situated usually near the optic disk or in the macular region, and vary in number from three to six or many more.

Instead of the miliary growth, a single large *tuberculous tumor* may appear and progress, producing the same destructive changes as a sar-

coma. It may be associated with a similar one in the brain.[1] According to Zur Nedden, the age of patients suffering from tuberculous tumor of the choroid has varied between one and a half and sixty-two years, although the age of childhood has furnished by far the greatest percentage. The condition must be differentiated from glioma of the retina in the young and sarcoma of the choroid in adults. The evolution of conglomerate tubercle of the choroid is usually more rapid than that of tumor. Scleral involvement and perforation generally occur early in the disease. Choroidal tuberculosis is rarely primary. Other signs of tuberculosis will usually be found in the general system. Occasionally in eye-grounds otherwise normal, and in patients in good health, isolated areas of choroidal atrophy with or without pigment heaping, or large patches of dense, slightly elevated pigment are seen, which probably represent healed tuberculous lesions—the so-called *obsolescent tubercles* or tuberculous areas.

Fig. 167.—Rupture of the choroid on the temporal side of the disk and pigmented traumatic choroiditis (see page 351) on the nasal side (from a patient under the care of Dr. Randall in the Children's Hospital).

Treatment.—Miliary tubercles of the choroid do not require any treatment directed to the eye itself, the vision of which may not be seriously affected. If a single large choroidal tumor is recognized, and the patient's general condition permits it, enucleation to avert general tuberculosis would seem to be a proper procedure. Instead of surgical procedures, injections of tuberculin (T. R.) have been employed by von Hippel and others with encouraging results, and should be given full trial (see also page 318).

Injuries of the Choroid.—Wounds of the Choroid.—Necessarily, in a perforating wound of the sclera, the choroid is also lacerated or

[1] *Chronic choroidal tuberculosis* is characterized by optic neuritis, optic atrophy, hemorrhages (tuberculous inflammation), and a diffuse, yellowish-white discoloration, occupying a considerable area of the eye-ground, within which are round, yellowish-white spots. Michel describes tuberculous granulation tumors of the choroid, which begin with the appearance of retinal detachment, and later cause abscess in the vitreous and shrinking of the eye.

incised, and no description other than that already given in this connection is required.

Foreign Bodies in the Choroid.—A penetrating foreign body may lodge in the choroid; the treatment described on page 293 is applicable.

Rupture of the Choroid.—The most important injury to which the choroid is subject, and which follows a blow upon the eye, is rupture. This generally manifests itself in a sickle-shaped crescent, commonly on the temporal side of the disk, rarely on the nasal side, and which very seldom extends in a horizontal direction. The rupture may be single or multiple, and sometimes is composed of several branches. The immediate effect of the blow is a hemorrhage preventing distinct observation. After its disappearance the fissure is evident to the ophthalmoscope as a yellowish-white stripe bordered with some disturbed pigment (Fig. 167).

The ruptures usually run concentrically with the papilla.

They may be either complete or incomplete, and may or may not be associated with breakage of the overlying retina. In rupture confined to the choroid, the retinal vessels pass over it. If the retina has also given way there is apt to be more hemorrhage than without such accident, and no retinal vessels are observed crossing the choroidal separation. Associated with choroidal rupture there may be a rupture of the sphincter of the iris (Duane and the author).

The ultimate effect on vision depends upon the size and situation of the rupture. At first there is very considerable disturbance of sight, partly due to effusion and partly to injury of the iris, sometimes associated with blood in the anterior chamber. This slowly clears away, and very good vision may result provided the change in the eye-ground has not been extensive. A deterioration of vision may occur a long time after such an injury owing to secondary changes in the optic nerve.

Treatment.—The pupil should be dilated with atropin; if there is much pain, a leech or two should be applied to the temple, a pressure bandage adjusted, and the patient put to bed. These measures suffice to encourage the absorption both of the blood and of the serous effusion.

Hemorrhage into the Choroid.—In the section on Unclassified Forms of Choroidal Disease variously shaped hemorrhages which appear in this membrane, and which by absorption give rise to atrophic spots, have been described. In like manner there may be hemorrhage from the choroid, the result of a blow. A choroidal hemorrhage may be distinguished from one situated in the retina by noticing the more diffuse character of the extravasation and the fact that the retinal vessels pass over it, but the diagnosis is difficult.

Detachment of the Choroid.—This is a comparatively rare clinical condition, although not infrequently found in enucleated, shrunken eyes. It may be spontaneous or traumatic, partial or complete. The detachment may be caused by blood, serum, a layer of lymph, or a new growth. Cases following cataract extraction, iridectomy for glaucoma, iridosclerectomy, and corneoscleral trephining (see page 634) are not very uncommon. The detached choroid protrudes as a dark mass into the vitreous, and the anterior chamber is shallow or obliterated.

It is caused by the passage of the aqueous humor through a rent in the attachment of the ciliary body beneath the choroid. The prognosis is favorable.

Meller, who has carefully studied the subject, classifies detachment of the choroid thus: Early postoperative detachment, innocent in character; late postoperative detachment, following trauma or occurring without cause; spontaneous detachment, either innocent, resembling postoperative separation, or malignant, causing blindness. If the retina is detached with the choroid the prognosis is most unfavorable.

Ossification of the Choroid.—This is occasionally found in eyes long blind and shrunken from destructive iridochoroiditis. The formation of bone occurs in the inflammatory tissue, and may be recognized by palpation in the form of an irregular plate, spicule, or complete shell. Calcareous degeneration is common in eyes of this character. The eyeball should be enucleated.

Atrophy of the eyeball is a condition characterized by diminution in the size of and alteration of the shape of the globe, caused by contraction of inflammatory exudations—for example, those formed in the uveal tract. It should be sharply distinguished, as Fuchs points out, from *phthisis bulbi*, which results from a suppurative inflammation (see page 357) and is associated with rupture of the sclera and partial evacuation of the ocular contents. The former may give rise to sympathetic irritation; the latter usually, but not always, is harmless.

Essential phthisis bulbi (*ophthalmomalacia*) is the name applied to a condition of the eye characterized by hypotony (softening) and diminution in its size which is unrelated to inflammation. There may be photophobia, pain, miosis, and partial ptosis. An intermittent variety has been described. It may follow injury and be connected with disease of the sympathetic.

CHAPTER XII

GLAUCOMA

Glaucoma is the name applied to several varieties of a disease of which increased intra-ocular tension is the most characteristic sign.

Varieties of Glaucoma.—Systematic writers are accustomed to divide glaucoma into (1) *primary glaucoma*, or that form which arises independently of clinically evident antecedent disease of the eye, and (2) *secondary glaucoma*, or that form which occurs as the sequel of a pre-existing ocular disease, often an inflammation of the uveal tract.

The primary variety of this disease has been divided into (1) *acute congestive glaucoma* (acute inflammatory glaucoma); (2) *subacute congestive glaucoma* ("glaucoma irritatif," chronic inflammatory or congestive glaucoma); (3) *chronic non-congestive glaucoma* (simple glaucoma, glaucoma simplex, chronic non-inflammatory glaucoma).

For clinical purposes it is convenient to retain these varieties of glaucoma and their descriptive names, but it should be distinctly remembered that in a certain sense the divisions are artificial, because an acute glaucoma may cease to have its congestive character and take on the signs which are ordinarily supposed to indicate the chronic variety of the disease, and the so-called glaucoma simplex may at any stage of its career develop symptoms of an acute progress, and lose its non-inflammatory or, more accurately, non-congestive character.

Symptoms.—The following is a syllabus of the symptoms common to the disease glaucoma, although all of these symptoms are not constantly present in each variety.

1. *Rise in intra-ocular tension*, or increased hardness of the eyeball. Formerly variations in tension of the ocular tissue, in part determined by the rise of intra-ocular pressure, were graded from T? ("stiffened sclera") to T + 3 ("stony hardness"). Intermediate degrees were expressed T + 1 and T + 2.

Since the introduction of satisfactory tonometers, particularly the instrument of Schiötz, the crude estimation of the impressibility of the eyeball by means of finger palpation has only an historic interest. The use of the tonometer is described on page 87; it measures the state of the tension of the ocular tissue, which is determined in part by the intra-ocular pressure.

2. *Change in the Size and Shape of the Pupil and Mobility of the Iris.*—The pupil may be round, oval or egg shaped, semidilated, or expanded to its fullest limit; the iris is sluggish in movement or entirely inactive. In simple glaucoma abnormal pupillary symptoms may be absent.

The pupillary space sometimes transmits a greenish reflex (hence the name given by the older writers) from the surface of the lens. The dila-

tation of the pupil is explained by paresis of the ciliary nerves or by constriction of the vessels of the iris. Partial atrophy of the lesser circle of the iris, which may lead to permanent dilatation of the pupil, is not uncommon after acute attacks of increased tension (Hirschberg).

3. *Loss of the Transparency of the Cornea.*—The cornea somewhat resembles the appearance of glass, the surface of which has been dulled by being breathed upon. This haziness is marked in the congestive types of glaucoma, but is absent or only slightly present in the simple varieties. If the cornea is carefully examined, the cloudiness will be found more decided in the center, and will resolve itself into very numerous closely aggregated points, the whole presenting a stippled or "needle-stuck" appearance. Iritis and iridochoroiditis may produce a similar appearance. The condition has been attributed to an edema of the cornea. Loss of corneal transparency with increased intra-ocular tension, such as may be caused, for example, by external pressure on the eye, is due, according to v. Fleishl, to the corneal fibers becoming doubly refracting.

4. *Change in the Depth of the Anterior Chamber.*—This symptom varies from an almost imperceptible shallowing to a complete obliteration. During the course of glaucoma the lens system and peripheral portion of the iris are pushed forward, and this causes the depreciation in the depth of the anterior chamber.

5. *Change in the Normal Appearance of the Iris and Turbidity of the Aqueous and Vitreous.*—The same edema which affects the cornea may also cause loss in the characteristic markings of the iris, so that its pattern becomes indistinct, especially in congestive forms of glaucoma. The veins of the iris may be dilated and tortuous; small hemorrhages are sometimes visible. Opacities in the media also are liable to form, and the lens itself may become cataractous.

6. *Alterations in the Conjunctival and Episcleral Vessels.*—In acute glaucoma there are usually general hyperemia and often edema of the conjunctiva, but in chronic inflammatory and sometimes even in simple glaucoma there are marked enlargement and tortuosity of the episcleral venous branches (see System II, page 49).

7. *The Excavation of the Nerve-head and the Surrounding Yellowish "Halo," or "Glaucomatous Ring."*—Under the influence of the increased intra-ocular pressure the most impressionable portion of the eye—the intra-ocular end of the optic nerve—gives way, and the glaucomatous cup is produced. According to Knies, congestion and edema of the nerve-head precede cupping, and according to Brailey and Edmunds, actual neuritis appears in advance of increased tension. The author and Gasparrini have seen glaucomatous excavation of the papilla follow retrobulbar neuritis. Wahlfors denies that increased intra-ocular tension alone is sufficient to cause cupping of the nerve-head, inasmuch as it requires a pressure of 135 mm. of mercury to produce such an excavation, and in glaucoma the rise rarely exceeds 100 mm. According to him, atrophy of the choroid is an important factor in this respect, because the resistance of the lamina cribrosa is thus reduced, owing to interference

with the vessel-bearing tracts which pass from the surrounding choroid into the nerve-trunk and branch in the anterior layers of the lamina. According to Schnabel, the excavation in the nerve-head in glaucoma is not due to increased intra-ocular tension, but to a form of degeneration of the optic nerve-fibers, which causes their complete disappearance and the formation of small cavities, both anterior and posterior to the lamina cribrosa (*cavernous atrophy of the optic nerve*). *Lacunar atrophy of the optic nerve* has also been observed in myopia without hypertony.

Axenfield has witnessed retrogression of a glaucomatous excavation, complete retrogression being rare, but the partial variety is not uncommon. This observation indicates that the excavation is due to increased intra-ocular pressure and that the position of the lamina cribrosa in glaucoma is not a constant one.

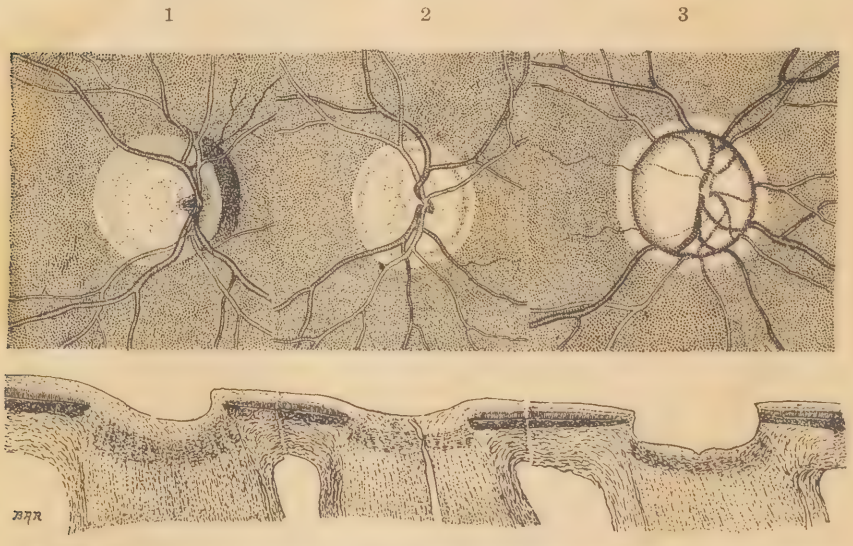


Fig. 168.—Excavations in nerve-head: 1, Physiologic; 2, atrophic; and 3, glaucomatous excavations (Randall).

The cupping of the optic disk is seen with the ophthalmoscope, and its depth is measured according to the directions given on page 107. It is also recognized by employing the *parallax test* with the indirect method as follows: The optic nerve is found in the usual manner by the inverted image, and the object lens moved from side to side. The entire eye-ground apparently moves with the motions of the lens, and the bottom of the excavation also seems to move in the same direction, but at a much slower rate. The contrast in the rate of the two movements is in a direct ratio with the depth of the excavation.

The cup varies from one beginning to be pathologic to a fully formed excavation. In the latter instance the excavation is complete to the scleral margin, and its edges are abrupt; the vessels are crowded to the

PLATE III.

The fundus of an eye with chronic glaucoma.

nasal side, bend sharply over the margin, and are lost to view behind the border of the cup, reappearing in fainter color at its bottom.

The papilla is encircled by a yellowish ring due to atrophy of the surrounding choroid.

It is important to distinguish between a large physiologic cup, an excavation due to atrophy of the optic nerve, and the glaucomatous cup. A physiologic excavation is partial and formed in a normally tinted nerve-head; an atrophic excavation is complete, shallow, and formed in a nerve-head of abnormal whiteness, owing to its loss of capillarity; and a glaucomatous excavation is complete, deep, and often of greenish hue. The microscopic appearances of a nerve-head containing a deep glaucoma cup are shown in Fig. 169 (consult also Fig. 168).

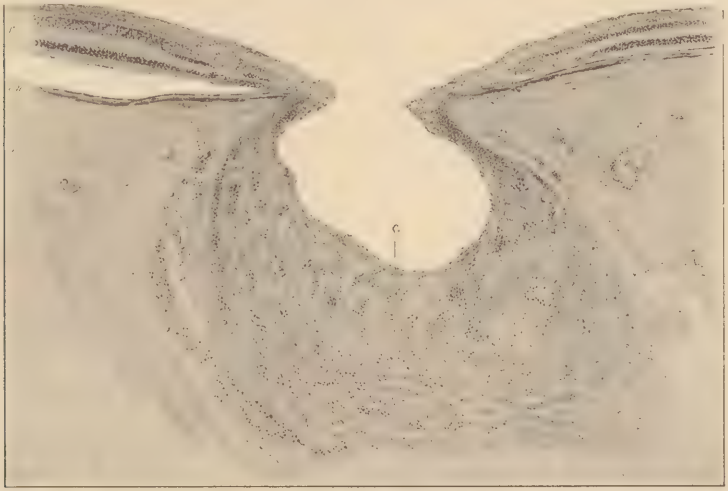


Fig. 169.—Section of optic nerve-head containing a deep glaucomatous excavation, the so-called kettle-shaped excavation: *r*, Retina; *ch*, choroid; *s*, sclera; *c*, cup, or excavation, pushing back lamina cribrosa.

The descriptions thus far given apply to typical forms of each variety of excavation. Sometimes it is a matter of considerable difficulty to decide between them, especially between an atrophic and a glaucomatous excavation where the latter is shallow; or between a physiologic excavation and glaucoma, where the former is associated with primary optic nerve atrophy. A diagnosis must then be based upon an examination of the other symptoms, particularly the field of vision.

8. *Vessel Pulsation on the Surface of the Disk.*—(*a*) *The Veins.*—There is often marked venous pulse, especially at the dark knuckles of the veins as they bend over the margin of the excavation, but this is a common ophthalmoscopic appearance in healthy eyes (see page 102), and hence cannot be utilized as a diagnostic symptom.

(*b*) *The Arteries.*—Pulsation of the arteries is a rare appearance except in aortic regurgitation, and therefore may be regarded as an important indication of increased intra-ocular tension, in high degrees of

which it is a striking symptom, the arterial trunks on the surface of the disk showing rapid alternate filling and collapse. It is usually, but not always, confined to the disk. The cause of spontaneous arterial pulsation resides in the resistance to the passage of the blood through the vessels, a resistance which, in turn, depends partly upon increased tension and partly upon spasmodic contraction of the vessels themselves. In cases of glaucoma in which this pulse is not spontaneously visible it may be induced by slight pressure upon the globe.

In addition to the *objective* signs of glaucoma just described, certain *subjective* symptoms are more or less constantly present.

1. *Pain.*—In acute attacks the pain is a severe neuralgia of the trigeminal distribution, and often, in violent congestive cases, an intense agony associated with great depression, pallor of the countenance, nausea and vomiting. In subacute attacks there is a less marked similarly located pain. In chronic cases there may be only a general feeling of discomfort, a sense of fulness, occasional shoots of neuralgia, or attacks described by the patient as "headache."

2. *Alteration in the Sensibility of the Cornea.*—Anesthesia of the cornea varies from a slight depreciation in its sensitiveness to an entire loss of sensation, as complete as that produced by cocain. Sometimes the anesthesia is not uniform over the surface of the cornea, but exists in spots or segments. It is due to the edema of the structure, which presses upon the filaments of the corneal nerves.

3. *Alterations in Central Visual Acuteness.*—This symptom varies considerably, and in chronic cases excellent sharpness of sight may be preserved for a long time. It is important to remember this, because it is not safe to depend upon central vision as a guide of the rate of progress of a chronic glaucoma. In each attack of subacute glaucoma the vision quickly fails, and gradually is recovered as the attack passes away. Each recurrence leaves a more permanent impression. In acute glaucoma a characteristic symptom is the sudden loss of vision, which in a few hours may be reduced to a light perception, and in certain malignant types rapidly becomes extinct.

4. *Alteration of the Refractive Power of the Eye and Diminution of the Amplitude of Accommodation.*—The former depends upon the change in the shape of the cornea, and the latter upon the effect of pressure upon the ciliary nerves. Alterations in the curvature of the cornea tend to produce an astigmatism "contrary to the rule" (see page 139), hence this is an important event in chronic glaucoma and in periods preceding its development. Diminished power of accommodation is evidenced by the desire of patients to change their reading-glasses to such as are stronger than the degree of refractive error or age of life would warrant.

5. *Alteration in Peripheral Vision, or the Field of Vision.*—A careful map of the field of vision in glaucoma is indispensable, and the restrictions present themselves in several forms: (*a*) The most usual and typical variety is partial or complete loss of the nasal field or of the upper or lower quadrant of the nasal side; (*b*) concentric restriction of the entire field; (*c*) restriction so constituted that the remaining field assumes an

oval or trowel shape; (*d*) sectional defects, often of the superonasal area; (*e*) loss of the entire field except a patch on the temporal side; (*f*) the formation of scotomas, which may be central, paracentral, annular, or peripheral (Figs. 170–178).

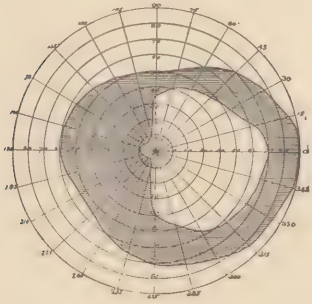


Fig. 170.—Field of vision of right eye in a case of subacute glaucoma. Loss of the nasal half and concentric restriction of the preserved field.

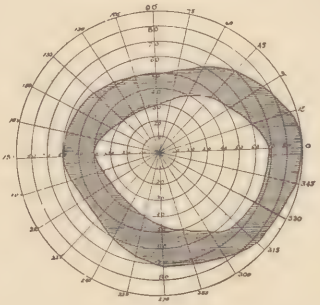


Fig. 171.—Field of vision of right eye in a case of chronic glaucoma, showing concentric restriction of the field.

The contraction of the color-fields is usually proportionate to that of the form-field, but this rule meets with exceptions. Under the influence of operative measures or miotics very decided improvement in the extent of the visual field may take place.

The tendency of the visual field is to contract progressively as the disease advances, and finally all portions are obliterated except a small

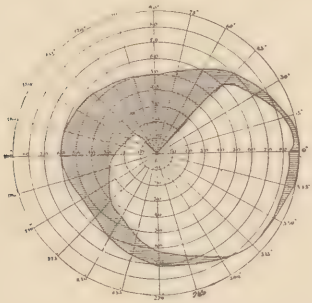


Fig. 172.—Field of vision in right eye in case of chronic glaucoma, showing sectional defect (superonasal quadrant).

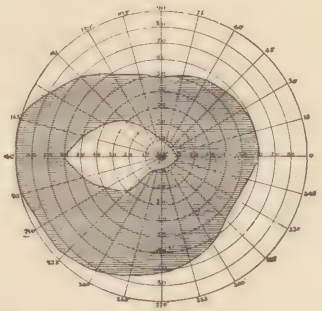


Fig. 173.—Field of vision of left eye in chronic glaucoma. Trowel-shaped patch preserved chiefly on the temporal side.

part upon the temporal side, which also disappears in the ultimate blindness (consult Fig. 174).

The contraction of the field of vision is an important index of the rate of progress in glaucoma, more important than depreciation of central vision; but it is not sufficient to trust to the periphery of the field for information. A search for *scotomas* is imperative. They must be found

either by the method suggested by Bjerrum (see page 81) or by ordinary perimetric methods, when care is taken to investigate each meridian and suitable test-objects are employed under varying degrees of illumination. Doubtless, as Bjerrum has pointed out, these scotomas are the result of the destruction of the fibers of the papilla at the margin or sides of the

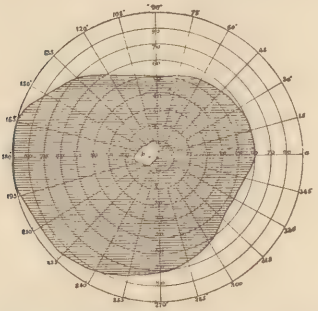


Fig. 174.—From the same case as Fig. 173, six months later; only a small patch of preserved field on the temporal side.

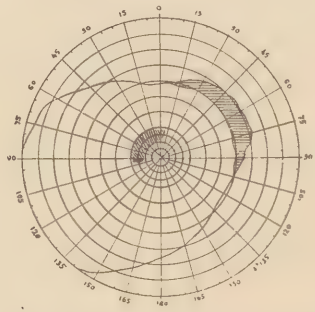


Fig. 175.—Just beginning contraction of nasal field; scotoma extending from blind-spot in a semicircular manner upward and inward.

excavation. They are topographically different from those which occur in simple optic-nerve atrophy, and may be utilized as a differential test between the two conditions, as Bjerrum has already suggested. This writer believes, and his observations have been confirmed by Meisling, Berry, Rönne, Sinclair, and other investigators, that a scotoma of this character is peculiar, in that wherever situated it is in direct continuity with the blind-spot. The scotoma may extend from the blind-spot and pass in an arc above and below the fixation point to end at the nasal side of it, about the horizontal meridian, or the defective area may merge with the blind area commonly existing in the nasal part of the field. This visual defect is often called *Bjerrum's symptom.* As Rönne points out, a small paracentral arciform scotoma is a common phenomenon, and to a break in the nasal field, whereby an alteration takes place in the boundary line of the field at the inner side from vertical to horizontal along the horizontal meridian, which it pursues for a varying distance before it again assumes a vertical or nearly vertical direction, he has given the name "nasal step." Central annular scotomas are demonstrable, and peripheral scotomas may be the forerunners of subsequent defects in the peripheral visual field (Figs. 177, 178). Scotomas may develop above and below the blind-spot and gradually coalesce with

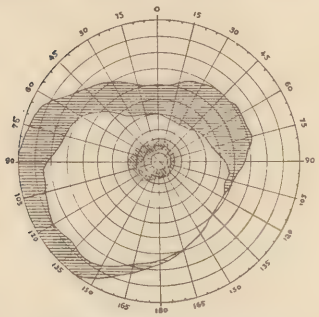


Fig. 176.—Annular scotoma in chronic glaucoma; moderate contraction of the peripheral field.

that spot, *i. e.*, a sickle-shaped scotoma is produced. Such scotomas, it is said, may be found in the field of an unaffected eye, its fellow being glaucomatous (Seidel's sign).

6. *Iridescent Vision.*—This consists of a definite ring surrounding artificial lights, which thus become invested with a colored halo ("halo vision"). First there is a dark band, followed by a concentric zone of prismatic colors, violet within and red without.

This phenomenon has been attributed to various causes depending upon physiologic or physical effects. Experimental evidence tends to support the opinion that the cause resides in the cornea, and depends on alterations in its epithelium, the result of exaggerated pressure.[1]

Subjective sensations of light are experienced at times by totally blind glaucomatous patients. The explanation is probably a mechanical one, and the sensation depends upon a dragging on the retina. In one

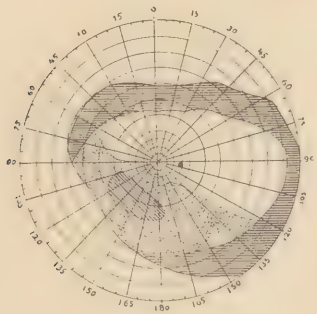


Fig. 177.—Visual field of right eye in chronic glaucoma, showing the mechanism of the loss of the lower and inner portion of the field, preceded by a scotoma, which gradually extends. Scotoma represented by parallel lines; area of dull vision which subsequently is completely lost, by dots.

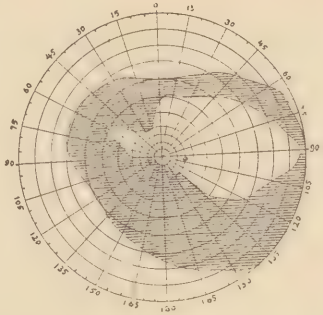


Fig. 178.—Later stage of Fig. 177. The scotoma has extended, and the area of the visual field in which sight was only dulled, and which is represented by dots in the preceding figure, has become completely dark.

case noted by the writer, both eyes being blind from glaucoma, the patient declared "all things seemed to be a sea of red fire."

The clinical varieties of glaucoma may now be described.

1. **Acute Glaucoma** (*Inflammatory or Congestive Glaucoma*).—This type of the disease is suitably divided into two stages:

(*a*) **Period of Incubation, or Prodromal Stage.**—This is characterized by sudden failure in the amplitude of accommodation, with a desire to resort to stronger reading-glasses; temporary obscurations of vision, either dim vision or quite complete loss of sight, lasting for many minutes; attacks of foggy vision, due to increased intra-ocular tension, all things apparently being invested with a haze; and the phenomena of colored halos around artificial lights. There may be some periorbital

[1] According to Schweigger, halo vision occurs in mild attacks of iritis with slight deterioration of vision. It may also be caused by a layer of mucus overspreading the cornea during chronic conjunctivitis. According to Myles Standish, the halo due to mucus has only the outer or red and yellow bands. The presence of blue in the halo may, therefore, be regarded as indicating increased intra-ocular tension.

pain, the pupil is slightly dilated, and the cornea and the aqueous humor faintly turbid. The appearance of the optic nerve at this stage is not characteristic.

These prodromes bear some relation to emotional excitement and insomnia, and may occur when the head is congested or after a full meal. After the eye regains its natural state, in a week or two the symptoms may reappear, again to subside and to be replaced by a fresh exacerbation or a true "glaucomatous attack." The incipient period of glaucoma may last one or more years.

(*b*) **Period of Attack, or the "Glaucomatous Attack."**—This commonly begins during the latter part of the night, sometimes preceded by prodromes, but sometimes without previous warning, and is characterized by violent pain in the head, so severe that it may induce nausea and vomiting. The face may be pallid, the extremities cold, or there may be flushing and general fever. The eyelids are swollen, the conjunctiva injected and sometimes chemotic, the cornea steamy and anesthetic, the pupil semidilated and motionless, the aqueous turbid, and the iris discolored. The tension rises very high, the tonometer registering 80 mm. of Hg and higher, and vision is rapidly lost, often only light-perception remaining, and even this may be abolished. Sometimes the attack is bilateral, or only a few hours elapse before the second eye is affected. Again, the interval between the two attacks may last weeks, months, or even years.

Gradually the symptoms pass away, with the exception of slight impairment in the mobility of the iris, some limitation of the field, and a little rise in tension. Blindness almost never occurs in the first onset. At this time characteristic ophthalmoscopic appearances are not present. After a number of attacks, examination of the eye-ground during a remission (the fundus is not visible during an attack) may reveal the characteristic cupping, the halo, and the arterial pulse.

If the disease is unchecked, the eye passes into a *glaucomatous state,* with fixed and dilated pupil, discolored iris, greenish reflex from the lens, vitreous opacities, shallow anterior chamber, and hazy cornea. Vision is now gradually destroyed and the eye reaches the *state of absolute glaucoma;* the ball is stony hard, the iris atrophic, the lens cataractous and pushed forward, the anterior chamber obliterated, the sclera discolored, the episcleral vessels coarsely injected, the cornea opaque, or perhaps ulcerated. Finally, there is disorganization of all the structures of the eyeball, and the sclera gives way with the formation of staphylomas, or the eyeball slowly atrophies as the result of choroiditis, change in the vitreous, and detachment of the retina. Acute glaucoma, instead of pursuing this course, occasionally passes into a chronic inflammatory type. *Spontaneous rupture* of a glaucomatous eyeball occasionally occurs; at the same time there may be choroidal hemorrhage.

Glaucoma fulminans is the name applied to an aggravated, rare form of the acute disease, in which the symptoms may be fully developed in a few hours without a prodromal stage. There is no remission, and the destruction of vision is swift and permanent.

2. **Subacute or Chronic Congestive Glaucoma.**—This type may or may not begin with the prodromal signs already described, or may be the sequel of repeated acute attacks. The eye gradually passes into a stage characterized by the constant presence of a series of symptoms which are often described under the title *chronic inflammatory* or *chronic congestive glaucoma.*

The cornea is deficient in transparency or positively steamy; there are marked tortuosity of the episcleral veins and some discoloration of the scleral tissue; the aqueous humor is turbid and the deeper media present opacities; ophthalmoscopic examination, when it is possible, reveals the cupped disk and pulsating vessels; the tension of the eye is raised; the pupil is semidilated, and the iris sometimes atrophic and sometimes not. Hence two types of chronic inflammatory glaucoma are described, one associated with degenerative changes in the iris and one without such association.

The field of vision is either contracted upon the nasal side or a quadrant of the field is darkened, or the other defects described on pages 373–375 may be discovered.

During the course of the disease acute or subacute attacks supervene; that is, there are sharp ciliary pain, increased steaminess of the cornea, increased injection of the eyeball, sinking of the vision, exaggeration of the tension, and marked anesthesia of the cornea. The attack gradually subsides, but in a few days or weeks repeats itself. Sometimes instead of a subacute attack of this character, an acute congestive exacerbation occurs, in all respects resembling the disease just described, and like it ending in absolute glaucoma or in degeneration of the tissues of the eye. This disease may last from several months to a year.

3. **Chronic Glaucoma or Non-inflammatory Glaucoma** (*Usually Known as Simple Chronic Glaucoma or Glaucoma Simplex*).—This type of the disease is characterized by an absence of the signs of glaucoma in the anterior aspect of the eye, at least on ordinary inspection. By careful examination, slight steaminess of the cornea may sometimes be detected, with a little lack of transparency in the aqueous humor, So, too, there may be some undue tortuosity of the perforating branches of the episcleral plexus. In general terms, however, there is an absence of congestive symptoms and there is no pain. The tension of the eyeball is always increased at some period of the disease, but this symptom is not constantly present, or it may be present at one portion of the day and not at another. Observations with the Schiötz tonometer indicate that increased tension is almost always present, although its degree may be a minor one. The depth of the anterior chamber is not materially altered. The chronic simple variety of glaucoma sometimes resembles chronic congestive glaucoma and vice versâ. If in the affected eye corneal involvement is made evident by nebulous vision, halos, etc., or, in other words, by irritative attacks, the case ceases to be one of simple glaucoma, and should be grouped with the chronic congestive types.

Almost without exception both eyes are affected simultaneously or successively; but it is difficult to fix the exact date of the onset of this variety of glaucoma, because of the absence of pronounced prodromal symptoms which precede the other types of this disease.

If both eyes are affected, the one is usually more advanced than the other, and the pupil is generally slightly larger on the side of the greater disease. In the later stages a greenish sheen from the pupil is often distinct. The central vision may be good, and in the earlier stages of the disease, after the correction of any refractive error, may reach the normal standard.

The media are clear, and the disease is detected with the ophthalmoscope by observing the characteristic cup in the nerve-head, the halo surrounding it, and the spontaneous arterial pulse, or its ready development by slight pressure.

The field of vision gives important information, and it assumes one or other of the characteristics described on page 372 (see Figs. 170–178). The central color perception is good, and the contraction of the peripheral color perception corresponds with that of the form-field.

Simple chronic glaucoma sometimes assumes a subacute or an acute nature like that already described, but often continues throughout its course without aggressive symptoms, having simply the characters of *optic-nerve atrophy with excavation*, from which it should be distinguished by the presence of increased tension and by the visual field phenomena which have been described (see page 373).

Causes.—(*a*) *Predisposing Causes.*—Primary glaucoma is rare before the fortieth year; not 1 per cent., according to Priestley Smith, begins earlier than the twentieth year. Glaucoma, generally unilateral, may occur in children, and *juvenile glaucoma* usually begins between the ages of fifteen and twenty; its prodromal period is of long duration. There may be a hereditary tendency to glaucoma, and, according to Nettleship, if the disease appears in more than one generation, it develops at an earlier age than in the preceding generation. Jews, Egyptians, and Brazilian negroes are said to be peculiarly liable to the disease. The glaucomatous eye is usually hyperopic, although Priestley Smith's statistics do not indicate a striking preponderance of this refractive state. There is a relation between smallness of the cornea and glaucoma. The average horizontal diameter of the normal cornea is 11.6 mm.; of the glaucomatous cornea, 11.1 mm. (P. Smith). Story's measurements yield an average of 11.9 mm. A large lens is a predisposing factor, and small eyes, in which the lens may be disproportionately large, are more liable to the disease than normal globes.

(*b*) *Exciting Causes.*—Glaucoma may be excited in eyes predisposed to the disease by worry, insomnia, bronchitis, cardiac disease, syphilis, gout, influenza, and neuralgia of the fifth nerve. Sometimes it follows injury and hemorrhage into the uveal tract, and *traumatic glaucoma* may be caused by a contusion of the globe without rupture of its coats. It has been stated that at corresponding ages usually there is higher average blood-pressure in glaucomatous subjects than in non-

glaucomatous subjects, and that arteriosclerosis and, therefore, increased blood-pressure are exciting causes of glaucoma. This contention is disputed by many observers, for example, H. Sattler, Freeland Fergus, H. C. Craggs, and C. G. Taylor. Overuse of ametropic or improperly corrected eyes, by causing uveal congestion, may bring on glaucoma in an eye predisposed to the disorder. The influence of strain upon the accommodation was explained by Snellen as follows: In the young eye, during accommodation for a near point, the diameter of the lens is reduced to about the same extent as that of the contracting ciliary muscle. The circumlental space remains about as wide as it was before, and the zonula remains tense as before. But the conditions are quite different in advanced life, when the elasticity of the lens is lost; the ciliary muscle contracts, but the form and size of the lens remain unchanged. The ciliary process is thereby pressed against the lens and the zonula slackened; hence the necessity of correction of refractive errors as a preventive measure. In a number of instances instillation of mydriatics has caused glaucoma. Acute glaucoma appears to be more frequent in winter than at other seasons of the year (Geisler).

Should a patient between his fiftieth and sixtieth year desire to change his reading-glass frequently, or use one stronger than is suited to his age or the condition of the refraction of his eye, there is reason to apprehend the onset of glaucoma. On the whole, the disease is slightly more common in women than in men. Those symptoms which have been described as prodromes are distinctive in themselves, and acquire an importance greater than any probable predisposition.

Pathogenesis and Pathology.—Three kinds of fluid are recognized within the eyeball: the blood within the blood-vessels, the lymph within the perivascular lymph-channels and the spaces in the uveal tract, and the intra-ocular fluid which is concerned with the nourishment of the vitreous and lens, supplies the aqueous chamber, and which, as was proved by Leber, proceeds from the epithelium of the ciliary body. It is probable that the lymph formation of the eye is produced in the ciliary body by a process of transudation, and not, as has been taught, by a process of secretion.

The chief stream of the intra-ocular fluid thus derived proceeds over the lens and through the pupil into the anterior chamber, traverses the latter to reach the angle formed by the junction of the iris and cornea, passes through the meshes of the ligamentum pectinatum, and by diffusion and filtration is taken up by Schlemm's canal. From this canal the greater quantity of the fluid passes into the anterior ciliary veins, a part of it being absorbed and eliminated by the iris (Nuel, Benoit). Only a very small portion of the fluid flows backward through the vitreous and escapes by way of the perivascular lymph-channels in the optic nerve. According to Priestley Smith, it is doubtful if there is any continuous stream from the vitreous into the aqueous chamber, but the anterior hyaloid membrane and suspensory ligament are easily permeated by it, and in health any excess of fluid in the vitreous chamber escapes by the filtration angle in the manner already described.

The pressure of the fluid regulates the outflow, so that when the afflux is increased a compensating increase of the efflux occurs (Snellen).

The fluids of the aqueous and vitreous chambers are nearly identical in composition and contain about 95 per cent. of water, 1 per cent. of extractives and salts, and a minute quantity of albumin. The intra-ocular pressure, which is equivalent to that of a column of mercury 25 mm. in height, is the same in the vitreous and the aqueous chambers, and preserves the shape and tension of the eyeball. If anything occurs to disturb its regulation, to quote Priestley Smith, "the pressure in the ocular chambers rises above the physiologic limits and we have the complex disturbance of function and structure called glaucoma."

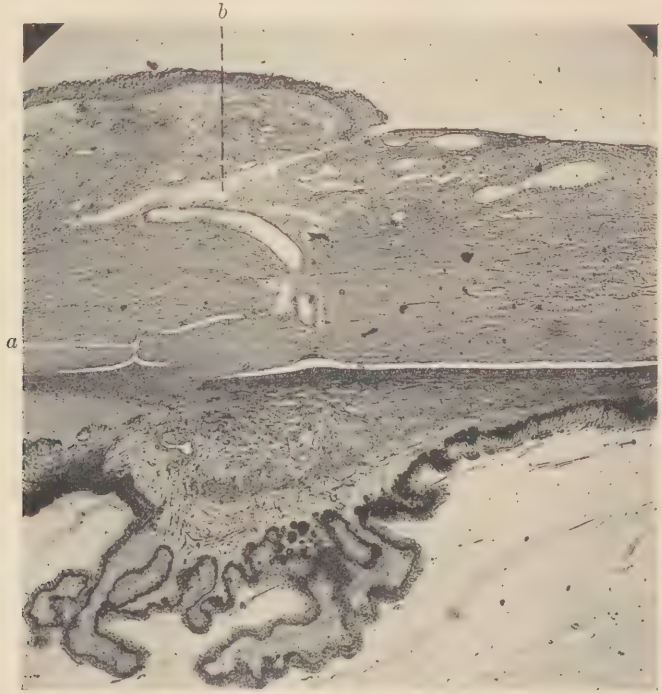


Fig. 179.—Photomicrograph of a specimen prepared by Dr. Brown Pusey. Communication between Schlemm's canal (*a*) and scleral vein (*b*).

What exactly are the factors potent in disturbing the regulation of pressure has never been satisfactorily determined, and numerous theories have been advanced.

The theory which has been and is still widely maintained is that one which assumes a diminution in the outflow and, therefore, a retention of fluid (*retention theory*). It obtained proper recognition when Knies and Weber demonstrated that in glaucomatous eyes, with shallow anterior chambers, there is an adhesion of the iris base to the periphery of the cornea, which prevents filtration at the angle of the anterior chamber and causes retention of the intra-ocular fluid (Figs. 180 and

181). This adhesion Knies regarded as an inflammatory process—that is, as a species of anterior iridocyclitis, while Weber considered it

Fig. 180.—Angle of the anterior chamber in a normal eye: *c*, Cornea; *s*, sclera; *g*, iris; *c.b*, ciliary body; *l.p*, ligamentum pectinatum; *s.c*, Schlemm's canal.

Fig. 181.—Angle of the anterior chamber in long-standing absolute glaucoma: *c*, Cornea; *s*, sclera; *j*, iris; *c.b*, ciliary body; *a.c*, angle of chamber closed by adhesive inflammation of the iris base to periphery of cornea, obliterating filtration area.

to be secondary to the pressure induced by an abnormally swollen ciliary body. The fact that a mydriatic does harm to an eye predisposed to glaucoma by dilating the pupil, rolling back the iris, and partly

closing the filtration angle, and that eserin does good by contracting the pupil and drawing away the iris from this angle, indicates, as Snellen has pointed out, that the explanation of glaucoma is to be found not in an increase of secretion, but in a disturbance of excretion.

Mydriatic glaucoma has also been ascribed to narrowing or closure of the iris crypts, an explanation which Priestley Smith declines to accept, for if it were correct, mydriasis should always be followed by rise of tension.

According to Priestley Smith, obstruction of the circumlental space —*i. e.*, the space between the margin of the lens and the surrounding structures—and consequent rise of pressure may follow increased size of the lens due to advancing years, unusual smallness of the ciliary area in hyperopia, or abnormal enlargement of the ciliary processes, and vascular disturbance which congests the uveal tract. It is possible that hypersecretion is sometimes concerned in the onset of glaucoma, and that serosity of the fluids plays a rôle in those forms which present a deep anterior chamber and wide filtration angle; but obstruction at this angle is part of the glaucomatous attack in the vast majority of cases.

C. Hess points out that bulbous outgrowths may develop on the summits of the ciliary processes in the course of life. Individual differences of the ciliary body are as important as differences in the size of the lens, and may have a distinct bearing on closure of the angle of the anterior chamber and the production of glaucoma.

Laqueur and other observers think that glaucoma depends upon obstruction of the intra-ocular lymphatics, which find their way out with the vasa vorticosa, owing to rigidity of the sclerotic coat. Brailey described a chronic inflammation of the ciliary processes and iris periphery, with distention of the vessels, as the earliest lesion in glaucoma. Stilling taught that a hardening of the sclerotic surrounding the papilla, through which waste fluid escapes, leads to glaucoma, and Strokousky attributes glaucoma to an *indurative scleritis*.

Evidently all retention theories assume that the cause of glaucoma depends upon an obstruction to the outflow of liquids from the eye occasioned by an interference with their escape through Fontana's spaces at the filtration angle, or through the perivascular lymph-channels in the posterior part of the eye, or through both of these exits; hence the fluid accumulates, the intra-ocular tension rises, and glaucoma results. In other words, upon the increased tension depend all the disturbances in the eye in this disease.

But this explanation is far from satisfactory to many observers, and certain objections have been advanced. For example, it is maintained that although adhesion of the root of the iris and blocking of Fontana's spaces are usually present in congestive glaucoma, it does not follow that this condition is the cause of the glaucomatous process; it may as well be, indeed, it is more likely to be, a result of it. Moreover, while it is practically always present in the eyes with long-standing glaucoma, it may be absent in an early stage of the disease. But especially is it true that this theory does not satisfactorily explain the

mechanism of so-called simple glaucoma, in which increased tension does not play a conspicuous rôle.

Wahlfors believes that the search for the cause of simple glaucoma must be made in the choroid, and maintains that the primary lesion is an atrophic process in the choriocapillaris, leading to nutritional disturbances in the layer of the rods and cones, whereby the important symptoms of this variety of the disease, diminution of light-sense, defects in the field of vision, and excavation of the nerve-head (see page 369), can be explained. He explains glaucomatous increase of tension by assuming that paralysis of the muscular network of the choroid causes a slowing of the intra-ocular liquids, that the retarded flow permits the deposition of formed elements in the channels of exit, and that, therefore, there is a retention of the liquid; finally, the venæ vorticosæ are compressed by reason of the increased tension, and venous stasis is the result. According to the manner and activity with which these factors influence the eye, the various types of glaucoma are produced.

Knies and other writers (see page 378), unable to reconcile any of the theories of glaucoma with the so-called simple variety of the disease, incline to separate it from the glaucoma class and place it among diseases of the optic nerve. In the present state of our knowledge, and keeping clearly in view the presence of increased tension, this does not seem advisable. Indeed, as Priestley Smith forcefully puts it, the nature of chronic and acute glaucoma is not essentially different, in that there is a close, though hidden, resemblance. "In both we find the predilection for small eyes; in both there is obstructive displacement of the iris—slowly established in the one case, rapidly in the other." Some authors explain this form of glaucoma by assuming a neuritis which blocks the lymph-channels in the optic nerve and its sheath, and which prevents the removal of effete matters which normally, to slight degree, occurs through these pathways, and thus causes increased tension and excavation. Hence the disease is sometimes called *posterior glaucoma*, to distinguish it from the other variety, *anterior glaucoma*.

Priestley Smith believes that "typical chronic glaucoma depends on slowly increasing contact of the iris with the cribriform ligament, arising through enlargement or advance of the lens, and involving in its early stages no serious compression of the iris."

W. Zimmerman considers that the primary cause of glaucoma depends upon a difference between the general blood-pressure and that of the eye. Parsons' experiments indicate that intra-ocular tension may *passively* respond to variations in the general blood-pressure, probably due to alterations in the volume of the intra-ocular blood-vessels, but it is uncertain that such passive changes are sufficient to account for glaucomatous attacks when these are apparently produced by excitement or emotion.

The notable increase of the amount of albumin which Uribe Troncoso has found in the aqueous humor of glaucomatous eyes induces him to

advance the theory that the symptoms which characterize glaucoma are best explained by its presence. The lesions in the blood-vessels which are found in glaucoma permit the passage of the albumin from the blood, and pathologic variations in the vitreous have also an important bearing on the glaucomatous process. Leber, however, who has reviewed Troncoso's work, has been unable to persuade himself that this author's views are correct. A. Knapp found in an eye with primary glaucoma an albuminous exudation which, by obliterating the anterior chamber through distention of the posterior chamber, produced the increased intra-ocular tension.

Brown Pusey, experimenting with the increase and decrease of intra-ocular tension which may be induced by varying osmotic pressures, believes that on them depends the explanation of the primary cause of glaucoma. M. H. Fischer holds that glaucoma is due to an edema of the eyeball, the amount of water contained in the hydrophylic colloids (chiefly the proteins) being increased by an augmentation of the quantity of acid present in the organ. The ordinary exciting causes of glaucoma are responsible for this abnormal production or accumulation of acid.

According to Thomas Henderson, the underlying predisposing and causal factor of glaucoma resides in a primary obstruction and closure of the pectinate ligament, or, as he prefers to call it, the cribriform ligament. This occlusion is the result of a sclerosis of the fibrous structure composing that filtration area which results, first, in a diminution, and, finally, in a complete obstruction of the outflow through it, leaving the iris, with its crypts, as the only efferent channel for the lymph-streams. While this sclerosis is the fundamental cause in all cases of glaucoma, he admits a second and variable agent, vasomotor in nature, which determines the acute attacks of increased intra-ocular tension. Henderson's theory does not seem to Priestley Smith to be sufficient. Levinsohn, finding a striking deposit of pigment cells, derived from the cells lining the ciliary processes, in an eye with absolute glaucoma, suggests that this may be an important factor in the production of acute glaucoma. Küschel believes that loss or disturbance of the elasticity of the supporting tissues of the eyeball is the cause of the various types of primary glaucoma. In all senile eyes this produces the "glaucomatous disposition."

Evidently all cases of glaucoma cannot be explained by any one theory, and the various clinical manifestations of the disease, as well as the results of treatment, indicate that sometimes one factor and sometimes another is the more potent in its activities. Of those which have been described, obstruction of the circumlental space dependent upon increasing size of the lens due to advancing years, obstructive displacement of the iris slowly or rapidly established (Priestley Smith), alteration in the composition of the intra-ocular fluid, blocking of the efferent channels by edema and exudation, sclerosis or pigment deposition, and vascular or vasomotor changes furnish the most satisfactory explanations of the various phases of glaucoma.

The *pathogenesis of secondary glaucoma* is easily understood, as the conditions which give rise to it—adherent leukoma, posterior synechiæ, swelling and dislocation of the crystalline lens, tumors and cysts at the angle of the anterior chamber—readily obstruct the outflow of the intra-ocular fluid and occasion its retention. Moreover, in serous cyclitis (uveitis), in addition to the accumulation of inflammatory cells in Fontana's spaces, there is an excess of secretion, highly charged with albumin, from the inflamed ciliary body. Intra-ocular tumors and hemorrhagic retinitis (thrombosis of the central retinal veins) tend to increase the pressure in the vitreous and thus cause secondary glaucoma.

As already pointed out, the apposition of the periphery of the iris to the cornea in primary glaucoma may at first be unassociated with inflammation; but if the apposition is long continued, proliferation of the endothelium of Descemet's membrane and the iris takes place and these two layers become adherent. Later the endothelium in large measure disappears, there is a round-celled infiltration of the deeper corneal layers and around Schlemm's canal, the tissue cells proliferate, and the iris becomes firmly bound down at its new position at the corneo-scleral junction. In the early stages of acute primary glaucoma the ciliary body and processes are engorged and swollen; later, and in long-standing cases, atrophy and shrinking occur. The changes in the choroid and their relation to the pathogenesis of the disease and the development of the excavation have been described. While there are no characteristic changes, as a rule, in the retina, in advanced cases atrophy of its elements are visible, and endo- and perivascular changes are evident, which may lead to hemorrhage. As already noted, edematous swelling and sometimes actual neuritis precede cupping of the nerve-head. Later there is backward depression of the lamina cribrosa and atrophy of the optic nerve-fibers. Alterations in the intrascleral passage of the venæ vorticosæ are sometimes discoverable, which depend upon proliferation of the endothelium in this position.

Diagnosis.—It is of the utmost importance that glaucoma shall be recognized, if possible, in its very incipiency. The most usual prodromal symptoms are a frequent desire to change the reading-glasses, periods of obscuration of vision, and the halos surrounding the lamp-lights.

The glaucomatous attack itself has frequently been mistaken for a "cold in the eye," for iritis,—and the disease has been aggravated by the instillation of atropin, which in almost all circumstances is contraindicated,—for neuralgia, and for reflex ocular pain. The condition of the pupil, the diminished depth of the anterior chamber, and the increased tension of the globe are the symptoms which should prevent so fatal an error. As pointed out by Parisotti and Trousseau, *ophthalmic migraine* sometimes simulates glaucoma, inasmuch as it may be associated with increased intra-ocular tension, arterial pulsation in the fundus, and contraction of the visual field.

The differential diagnosis of simple chronic glaucoma and atrophy of the optic nerve has been referred to and presents considerable diffi-

culty. The absence of constant increased tension in the simple form of the disease, or at least its doubtful presence, removes an important diagnostic point. Examination with the tonometer is of great importance in these circumstances. Help may be obtained by observing the visual fields. In glaucoma the color-fields present a restriction corresponding with that of the form-fields, while in atrophy the peripheral color vision, especially for red and green, is markedly deficient. The diagnostic value of the shape of the field, and especially of the scotomas, notably *Bjerrum's scotoma*, has been described (see page 374).

Examination of the light-sense is important. In glaucoma the "light minimum" is said to be deficient, but the "light difference" not far from normal; in pure optic-nerve atrophy there is imperfect ability to distinguish between different intensities of illumination ("light difference"). In other words, according to Samelsohn, the light-perception power in glaucoma is much lessened, while the light-difference power is relatively not greatly interfered with; in optic-nerve atrophy the reverse is usually the case. Wahlfors, confirming observations made long ago by Mauthner and Förster, insists that reduction of the light-sense is one of the most frequent symptoms of simple glaucoma, and that night-blindness may first call the patient's attention to his eyes. Moreover, this reduced light-sense may exist for years before the real nature of the disease is evident.

It is an inexcusable error to confound the failing vision of chronic glaucoma with that of cataract, the greenish reflex of the lens, which may be seen in the pupillary space, being mistaken for an opacity of the lens. Eyes have been permitted to pass into blindness, and their possessors deluded with the hope that they were waiting for the ripening of a cataract which never existed. An ophthalmoscopic examination would settle the diagnosis at once.

Prognosis.—Glaucoma does not tend to spontaneous cure, but, if unchecked, to blindness; hence the prognosis is unfavorable if proper treatment cannot be applied. Prognosis also depends upon the type of the disease and the stage of its development. Other things being equal, uncomplicated acute cases furnish the most reasonable hope of complete cure, and if a technically correct operation can be performed *early*, the result is usually satisfactory. In chronic cases much depends upon the amount of degenerative change in the tissues, and the prognosis must be guided by the state of vision, the extent of the field, and the condition of the iris. The effect of treatment upon the progress of glaucoma is included in the following section:

Treatment.—In the majority of cases of glaucoma an operation is needed to check the disease.

It may happen, however, that an operation is not at once possible or advisable, and hence the miotics should be quickly and thoroughly used. In the prodromal stage eserin salicylate or sulphate should be employed and will usually relieve the symptoms. In acute cases eserin, in a strength of from 1 to 4 grains (0.065–0.26 gm.) to the ounce (30 c.c.), acts favorably, and often with surprising rapidity, provided the pupil will

contract under its influence. Pilocarpin hydrochlorate, 2 to 5 grains (0.13–0.324 gm.) to the ounce (30 c.c.), may be substituted. Miotics act by drawing the iris away from the filtration angle, and, by contracting the pupil, cause widening of the spaces of Fontana and absorption of the fluid; also iris-surface filtration is increased. A drop or two of the selected solution should be instilled every hour or two until relief is obtained; if this does not occur promptly, iridectomy or one of its substitutes should be performed. Arecolin in 0.5 per cent. solution has also been used; with this drug the author has had no experience. Dionin and adrenalin chlorid have been much employed in the treatment of acute glaucoma. The former (in 5 per cent. solution) often acts efficiently as a lymphagogue and analgesic; the latter (1 : 10,000) must be used with caution, as occasionally it increases the intra-ocular tension. It may be added to the solution containing the miotic.

In addition to the use of eserin or pilocarpin during an acute attack the temple may be leeched, warm fomentation applied, and rest and relief from pain secured by the exhibition of morphin and chloral, the latter drug having some influence in reducing tension. Full doses of salicylate of sodium, however, act more favorably than any other constitutional remedy (Sutphen, Friedenwald); indeed, they are most useful in any form of glaucoma associated with pain. An interesting observation of Morax is that reduction of tension in glaucomatous eyes in syphilitic subjects has followed the injection of salvarsan. To lower intra-ocular tension Fischer and Thomas recommend subconjunctival injections of *sodium citrate* (4.05–5.41 per cent. solution). Of these solutions, 5 to 15 minims (0.30–0.92 c.c.) are injected, and if the injections are frequently employed, the weaker of the two, diluted with 2 to 4 parts of physiologic salt, is employed.

In chronic inflammatory (subacute) glaucoma, eserin (the sulphate or salicylate) or pilocarpin should be employed until it is decided what operation shall be done and when it shall be performed. The other remedies advised in the preceding paragraphs are also useful and should be used.

There is much difference of opinion in regard to the value of miotics in the treatment of chronic, non-congestive glaucoma. In the opinion of some surgeons who deprecate operation in this form of glaucoma, they represent the chief therapeutic measure, while in the opinion of others they are practically without value. Neither of these extreme views is correct. That miotics can hold the disease in check for long periods of time cannot be doubted. They must be properly used, that is, the pupil must be kept contracted. For this purpose Posey prefers salicylate of eserin, beginning with a solution of $\frac{1}{10}$ grain (0.00648 gm.) to the ounce (30 c.c.), and gradually increasing the strength until, if the drug has continued to act favorably, at the end of three years the solution has a strength of 3 grains (0.195 gm.) to the ounce (30 c.c.). The author prefers pilocarpin, as it is equally efficient and less irritating; the strength should usually be twice that of the eserin solution. Conjunctival irritation can generally be prevented if the solutions are always

fresh and sterile, and if the conjunctival sac is frequently irrigated with a boric acid lotion.

Massage of the eyeball is of distinct advantage; it may be followed by improvement in vision and deepening of the anterior chamber. Apparently it assists the action of the miotics. Usually it is not possible to employ it in acute glaucoma, but in simple glaucoma and in eyes with mild attacks it certainly temporarily lowers the tension. It may be applied by means of various instruments (see page 261), that is, vibration massage or with the help of suction cup (suction massage). Just as good results can be obtained by simple massage with the fingertips. Unfortunately, the value of massage has been unduly magnified by irregular practitioners. It is useful only as an adjuvant to other well-recognized procedures, operative and medicinal. Strychnin and nitroglycerin should be given to patients with chronic glaucoma, especially the latter drug if there is increased vascular pressure. *High-frequency currents* have been advised.

Iridectomy, in the author's opinion, in most circumstances, continues to be the most satisfactory operative procedure in the treatment of *acute* glaucoma. It should be performed *early*, in the prodromal stage if possible, while the excretory apparatus is still intact and *before the root of the iris is welded to the cornea*. General anesthesia should be induced before its performance, because the high tension of the eyeball somewhat nullifies the action of cocain. Much depends upon the exact position of the iridectomy, which is difficult of performance on account of the narrow anterior chamber, and no caution should be omitted which will secure perfect quiet on the part of the patient.

In performing iridectomy for the relief of acute glaucoma the following directions should be borne in mind: If a keratome (Fig. 300) is employed, it should be entered through the sclerotic coat 2 mm. from the apparent border of the cornea, and, after the completion of the incision (see page 624), should be slowly withdrawn in order to prevent a sudden gush of aqueous humor, and a too rapid reduction of tension, which might be followed by intra-ocular hemorrhage. If the anterior chamber is shallow the iridectomy is usually more easily performed, and with better results, if a Graefe cataract knife (see Fig. 345) is employed in the usual manner (see page 655). The excision of the piece of iris should be complete up to the periphery—*i. e.*, up to the ciliary border—and no portion of the excised iris must remain in the angles of the wound (compare with page 390). This is a much more important matter than the excision of a large piece of the iris—for example, one-fifth of it—as is usually advised. A comparatively narrow technically correct iridectomy yields satisfactory results. If the tension is very high, preliminary scleral puncture, as advised by Priestley Smith and Gifford, is a useful procedure. It should be remembered that scleral puncture has been followed by intra-ocular hemorrhage (A. Knapp).

A favorable result may be expected from iridectomy if the tension is lowered; an unfavorable one if this remains high. If there is a sudden

rise of tension a short time after the operation, accompanied by severe pain, there is reason to suspect intra-ocular hemorrhage.

Section of the iris is often followed by an extensive hemorrhage into the anterior chamber. A prolonged effort to get rid of this blood should not be made lest the trituration produce cataract. The blood will absorb, although it may take many days and even weeks before this is entirely accomplished.

The re-forming of the anterior chamber is sometimes delayed as long as a week. Occasionally a day or two after the operation there is some slight rise of tension in the eye, which is of temporary character.

There is difference of opinion as to whether the eye should be bandaged or not after operations of this character. The author believes that not only should a bandage be applied for the first few days to the eye upon which the operation has been performed, but also to the fellow eye; and that the one placed upon the affected organ should remain there until complete restoration of the anterior chamber has taken place. The eye which has not been operated upon should be kept thoroughly under the influence of eserin or pilocarpin during the course of the treatment, because it is well known, in acute glaucoma, that iridectomy may be followed by a speedy outbreak of the same disease in the opposite eye. In most instances the iridectomy should be placed directly upward, so that the overhanging upper lid may cover the coloboma. Should the primary iridectomy fail to reduce the tension permanently it may be necessary to reinforce it with a cyclodialysis or an anterior sclerotomy (see page 635).

The author has expressed his faith in the efficacy of iridectomy in *acute* glaucoma. Col. Elliot and other surgeons decide in favor of corneoscleral trephining or other operations designed to establish permanent filtration in the *acute* as well as in the *chronic* forms of glaucoma.

If the eye which has not been subjected to operation has a decidedly shallow anterior chamber, and if there is a history of prodromal glaucomatous phenomena, it should be submitted to operation as soon as the iridectomy wound in the opposite eye has firmly healed, certainly before the patient passes from skilled observation, because it is practically certain that it will be attacked like its fellow. If the signs of impending glaucoma are not clear and the eye is nevertheless suspected, the mydriatic test suggested by Edward Jackson, Harlan, and Brailey, which consists of the instillation of a solution of homatropin and noting whether it produces any rise in intra-ocular tension or pulsation of the vessels of the fundus, may be employed. Should the test be positive, it would seem proper to perform at once what Treacher Collins has called a *preventive iridectomy*, or, following Col. Elliot's advice, a corneoscleral trephining. If this is declined or deemed inadvisable, the patient should use daily a solution of a miotic (strong enough to keep the pupil contracted), and be provided with a stronger solution to be used in an emergency—*i. e.*, during a sudden attack. If both eyes are affected, both should be operated upon, pro-

vided the conditions are suitable, at proper intervals; sometimes in acute cases operation on one eye must immediately be followed by operation on the other.

One of the complications which may follow the operation of iridectomy in glaucoma is the formation of a bulging scar at the seat of the incision, sometimes called a *cystoid cicatrix*. This is especially true if due care has not been taken to free the angles of the wound from adherent iris. On the other hand, in severe cases, this very cystoid cicatrix, by permitting a filtering of the liquids, has been regarded as a favorable condition. In this connection the modern operations for the relief of glaucoma, by means of which a *filtering area* is produced, must be considered. They are discussed on page 629.

The treatment of chronic inflammatory (subacute) glaucoma is less likely to be followed by the brilliant results seen in acute cases; and many instances are on record in which after the performance of an operation, entirely correct in its technic, the disease has not been stayed, or malignant glaucoma (see page 390) has resulted. This is particularly true if degenerative changes have occurred in the iris. Nevertheless, iridectomy preceded, if necessary, by sclerotomy or one of its substitutes, that is, corneoscleral trephining or iridosclerectomy, will more often yield a satisfactory result than if medicinal measures are alone relied upon, and should be performed as early as possible.

There is much difference of opinion in regard to the value of iridectomy in simple glaucoma (chronic non-inflammatory glaucoma), and some surgeons doubt the propriety of its performance in this disease, and depend upon miotics and certain internal remedies—for example, strychnin and nitroglycerin. Statistical information indicates that in a limited number of the cases of simple glaucoma submitted to iridectomy the results are immediately unfavorable—that is, the disease is not only not checked, but rapidly progresses to blindness; in a fair percentage of cases (15–45 per cent.) the disease remains stationary—that is, the iridectomy maintains the condition of vision which was present before the operation; in a certain number of cases there is temporary amelioration, but later slow advance of the disease—a rate of advance, however, that is slower than if operation had not been performed; in a comparatively small percentage of cases the operation is followed by perceptible and permanent improvement in vision.

In a certain number of cases (Friedenwald has collected 24, 18 of them being women) a perfectly smooth iridectomy is followed by *malignant glaucoma*. The symptoms which usually appear one or two days after the iridectomy are: marked increase in tension, obliteration of the anterior chamber, fixation of the coloboma, ciliary tenderness, chemosis of the conjunctiva, swelling of the lids, and rapid loss of vision. Hence the importance of following Schweigger's advice to operate in chronic glaucoma affecting both eyes, first upon the one with the more advanced disease, even if it is blind. If no complication arises, there is reason to hope that iridectomy on the fellow eye will be followed by a normal healing process. The treatment of malignant glaucoma consists in the

instillation of eserin, or posterior sclerotomy, and the administration of large doses of salicylate of sodium (Friedenwald).

It is not entirely certain how iridectomy cures glaucoma. It has been suggested that this is accomplished by the removal of the portion of tissue which closes the angle at the anterior chamber; by the moderation of the blood-pressure in the iris (Exner); by the filtration of the fluids of the eye through the line of healing, which, for this reason, has been called the *filtration scar;* by the permanent drain which the cut surface of the iris affords, inasmuch as it is not closed by reparative processes (Henderson). The details of performing iridectomy and sclerotomy will be described in the chapter devoted to Operations.

The operation of *sclerotomy* has been used as a substitute for iridectomy, but the weight of testimony in favor of the latter operation is sufficiently great not to make it a more desirable mode of procedure than iridectomy except in selected cases. Every iridectomy which is peripherally situated, and in which the knife enters through the sclera some distance from the apparent border of the cornea, is in itself a sclerotomy. It is useful as a supplement to iridectomy if the tension is not reduced, and may be employed in old blind glaucomatous eyes to relieve pain. According to the late Dr. de Wecker, sclerotomy, followed later by iridectomy, which can then be performed more correctly owing to the improved state of the eye, is preferable to a primary iridectomy.

In recent years a number of important and usually satisfactory operations have been devised for the purpose of maintaining a *filtering cicatrix* or *area,* and, in so far as our present knowledge enables an opinion to be formed, their permanent results, other things being equal, are much more satisfactory than the simple iridectomy of former times. Of these, the most important are *Lagrange's operation,* by means of which an iris-free filtering cicatrix is produced with a combined iridectomy and sclerectomy (see page 629), *Herbert's wedge-isolation operation* (see page 630), *trephining the corneoscleral border* (Fergus-Elliot operation, see page 632), and Holth's *punch operation.* The value of Heine's operation, or *cyclodialysis* (see page 635), in the treatment of glaucoma has been much discussed in the past few years; but it is evident that while it has its uses it cannot replace iridectomy or the operations just referred to.

The operation of *sympathectomy,* or excision of the superior cervical ganglion of the sympathetic, for the relief of glaucoma has been performed a number of times. Little is heard of the operation at the present time, and evidently it has, very properly, failed to secure a permanent place in ophthalmic surgery.

For the relief of the pain of absolute glaucoma *opticociliary neurotomy* has been performed, and is still advocated by some surgeons. In the opinion of the author, enucleation or one of its substitutes is a better operation, but he also has had some excellent results with cyclodialysis and from corneoscleral trephining. In these circumstances, if the other eye shows any prodromal signs of glaucoma, it would seem proper that

an iridectomy or a corneoscleral trephining should be performed in anticipation of the glaucomatous attack.

Secondary glaucoma, or that form which arises in consequence of some pre-existing disease of the eye, may, like the primary variety, assume an acute or chronic type.

It may follow inflammation of the iris and ciliary body with the production of extensive synechiæ; serous cyclitis, ulcers of the cornea which have perforated and produced adherent cicatrices or staphylomatous bulging; swelling of the crystalline lens after needling; discission of after-cataract and, occasionally, primary extraction of cataract; dislocation of the lens; detachment of the retina, associated with severe hemorrhage; the growth of a choroidal sarcoma or other intra-ocular tumor; and choroidoretinitis, plastic choroiditis, or disease of the retinal vessels. Owing to injury the vitreous may pass forward into the anterior chamber, block the channels of exit, or press against the iris, and thus cause secondary glaucoma. Adhesion of the lens capsule to the cornea following traumatism or cataract extraction not infrequently results in secondary glaucoma.

In most of the instances mentioned there is no difficulty in diagnosticating secondary glaucoma by the history of the case and the knowledge of the pre-existing disease. This is not so easy if the original trouble has been deep in the eye, for example, a sarcoma. In these cases the glaucoma is usually absolute.

Treatment.—Secondary glaucoma, in general terms, requires the same treatment as the primary form of the disease, which must be modified according to the surrounding ocular conditions. A dislocated lens, or a lens swollen after discission for cataract, should be removed. Absolute glaucoma associated with great pain, if there is any suspicion of intra-ocular growth, indicates excision of the globe.

Hemorrhagic glaucoma is one type of secondary glaucoma in which numerous retinal hemorrhages appear as the result of thrombosis of the retinal vessels, or hyaline degeneration of their walls, or other causes likely to produce extravasation of blood (albuminuric retinitis). The tension rises and the character of the disease may be acute, subacute, or chronic. This condition should be sharply differentiated from primary glaucoma associated with retinal hemorrhages, although sometimes it is exceedingly difficult to decide whether the glaucoma is secondary to the hemorrhages, or whether the hemorrhages have been produced by alterations in the tension of a glaucomatous eye. With the ophthalmoscope one may see the ordinary appearances of glaucoma and numerous retinal hemorrhages; or, in addition, there may be the lesions of the disease which has caused the hemorrhages and the glaucoma which followed them. Hemorrhage into the vitreous may occur, obliterating the fundus reflex; the cornea is steamy, the anterior chamber obliterated, the iris discolored, and the eyeball intensely injected and very hard.

Iridectomy is not usually followed by good results in hemorrhagic glaucoma. It may lead to permanent blindness by fresh hemorrhagic exacerbations. If attempted it should be preceded by posterior scle-

rotomy. Corneoscleral trephining has been tried. The results of anterior sclerotomy are more favorable than those of simple iridectomy. Tapping the vitreous alone may be followed by relief, and cautious paracentesis of the anterior chamber was advocated by Bull. If the pain becomes intense and blindness ensues, enucleation is required. General treatment is of importance, as the patients are usually the subjects of vascular disease and high arterial tension: the cautious use of cardiac sedatives, nitroglycerin, and strict regulation of the diet and mode of life. Locally, measures to relieve ocular congestion and the miotics may be employed; dionin and holocain often afford decided relief.

Complicated Glaucoma.—Two kinds of complicated glaucoma are described which may be looked upon as varieties of the secondary form of the disease, namely, *cataract with glaucoma* and *high myopia with glaucoma*. In the former condition one eye alone is usually affected. It is to be distinguished from the lenticular opacity produced by absolute glaucoma. During the formation of cataract glaucoma may occur, due probably to swelling of the lens and lessening of the circumlental space, and it is important to test frequently the intra-ocular tension of patients with developing cataract; preliminary iridectomy may be required.

In high myopia with glaucoma the usual changes in the field of vision and the papilla are present. In addition to this there is more or less choroidal disturbance, which may itself be the cause of the glaucomatous condition. According to Hotta, the first change produced by the increased tension is an excavation of the nerve-head, and subsequently an ectasia into the intervaginal space of the immediately surrounding sclera.

Hydrophthalmos, or that disease of the eye which has been looked upon as a congenital glaucoma, has been described on page 280.

CHAPTER XIII

DISEASES OF THE CRYSTALLINE LENS

Congenital Anomalies.—In addition to congenital cataract and congenital displacement of the lens, which are described on pages 411 and 412, two anomalies require mention:

1. **Coloboma of the Lens.**—This defect occurs usually with a similar defect in the iris and choroid. The normal, rounded margin of the lens is replaced by a straight margin in a horizontal direction or incurved. The amount of the defect varies from a slight indentation to about one-quarter of the lens substance. It is almost always situated in the inferior half of the lens.

2. **Lenticonus.**—This is an abnormal curvature of the posterior surface of the lens or an anomaly of the nucleus (L. Müller), either unilateral and associated with lenticular opacities, or without such association, and then usually bilateral. Anterior lenticonus also occurs. With the plane mirror a sharp red disk, surrounded by dark shadows, like an oil-globule in water, may be seen (Knapp). *Congenital aphakia*, in association with faulty development of the anterior part of the globe, has been reported; it may also result from the absorption and degeneration of a previously formed lens.

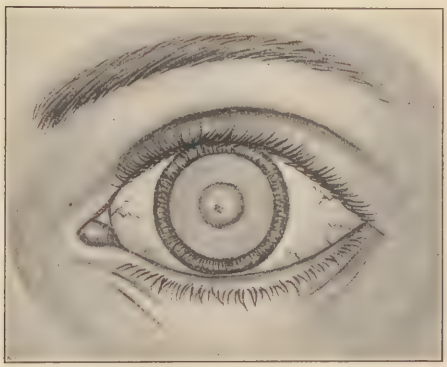


Fig. 182.—Posterior lenticonus (from a patient in the University Hospital).

Cataract.—Under the term *cataract* are included several types of an opaque condition of the crystalline lens, of its capsule, or of both these structures, which anatomically are distinguished by the titles *lenticular*, *capsular*, and *capsulolenticular*.

Varieties of Cataract.—(1) *Primary;* (2) *secondary* to disorders in other portions of the eye; (3) *symptomatic* of a general malady or local injury.

A cataract is either *partial* and stationary, or progressive and becomes *complete*, and clinically is classified as *senile*, subdivided into *nuclear* and *cortical;* or, according to Axenfeld, *subcapsular, supranuclear,* and *nuclear; juvenile* or *presenile; congenital*, subdivided into *complete* or *partial; secondary* or *complicated; traumatic;* and *after-cataract.*

Cataracts are also classified according to their consistence as *hard*, *soft*, or *fluid*, and sometimes are designated by their color as *black*, *white*, *amber*, etc. Although in many instances the precise division of cataract into special varieties may be unimportant, the following table, compiled from the classifications employed in various standard works, may be useful to the student as a résumé of what has gone before:

- **Anatomically**
  1. Lenticular.
  2. Capsular.
  3. Capsulolenticular.
- **Clinically**
  1. Senile — (*a*) cortical, (*b*) nuclear — general.
  2. Juvenile or presenile.
  3. Congenital
     - (*a*) complete — complete; congenital.
     - (*b*) partial — lamellar, or zonular; axial; pyramidal, or polar.
  4. Complicated or secondary — anterior polar cataract; posterior polar cataract; complete cataract.
  5. Traumatic.
  6. After-cataract.

Symptoms.—The following symptoms are present with more or less constancy in cataract, exemplified by the senile form of this disease:

1. *Change in Visual Acuteness.*—The amount of depreciation of sight depends upon the situation and extent of the opacity, and sometimes upon alterations in the refractive power of the lens. Thus there may be an increased refraction at the nucleus, causing myopia, often called *prodromal myopia*. In these circumstances distant vision is improved by concave lenses and reading becomes possible without the aid of convex glasses. This is the so-called "second sight," which in itself is strong presumptive evidence of the existence of cataract. In like manner the change in the lens may produce an irregular astigmatism, or an astigmatism "against the rule" may develop.

2. *Hyperemia of the Conjunctiva.*—This is caused by the strain which the effort to see through a somewhat clouded lens produces.

3. *Pain and Photophobia.*—These symptoms are not prominent; but sometimes, owing to the condition of disturbed choroid which commonly is associated with cataract, patients complain of dull, aching pain or other asthenopic symptoms. Tinted glasses relieve the photophobia and permit slight dilatation of the pupil, which sometimes improves vision if the opacity is central. Pain, with rise of tension on account of swelling of the lens, occasionally occurs. Indeed, *acute glaucoma* may be caused by this swelling of the lens during the formation of cataract, and the state of the intra-ocular tension deserves close attention in all patients with formed or forming cataract.

4. *Polyopia* and *monocular diplopia* are occasionally the result of incipient cataract, and are due to the irregular astigmatism which the alterations in the lens have produced.

5. *The Anterior Chamber.*—This may be normal in depth—the usual

condition; shallower than normal—indicating a swollen lens; or abnormally deep—a symptom of a small lens.

6. *The Pupil.*—This may be natural in appearance and the mobility of the iris entirely normal; but sometimes the effect of exclusion of light or of a mydriatic fails to induce a dilatation of the pupil.

We speak of the "color of the pupil," and this varies in cataract according to the degree of maturity and the hue of the opacity. Hence in the unilluminated pupil no change is seen in its color in incipient cataract; but in a ripe cataract the pupillary space may appear dull, gray, and even white, according to circumstances. In examples of so-called "black cataract" the pupil is dark. The mere inspection of the pupil, however, without optical aid is not sufficient to ascertain the condition of the lens, which continues to increase in size even with advancing years, if it remains clear (Priestley Smith). But it becomes firmer, straw colored, and reflects more light. This creates a dull sheen in the pupil which may be mistaken for cataract. The yellowish tint of the lens in advanced life may modify the relative perception of colors, which, indeed, may be so pronounced as to create *blue-blindness* (C. Hess).

Diagnosis.—From what has been said, it is apparent that the absolute diagnosis of cataract depends upon the use of the ophthalmoscope. Since the introduction of the ophthalmoscope, the *catoptric test* has fallen into disuse, although it may be employed to determine the presence of the lens and in the diagnosis of black cataract.

This test is performed as follows: If, in a dark room, a lighted candle be moved before a healthy eye with dilated pupil, three images of the flame will be seen: two erect, formed by reflection from the convex cornea and anterior surface of the lens, the former producing the bright image and the latter the more diffuse; and one inverted, relatively clearer, from the posterior surface of the lens. If, now, the lens be opaque, the inverted image will be wanting, the deeper erect image also disappearing when the opacity involves the capsule, the corneal image being then alone visible.

Before using the ophthalmoscope for the detection of cataract the pupil should be dilated, preferably with homatropin, cocain, or euphthalmin. The examiner proceeds in the manner described on page 99, and will detect in incipient cataract spots or streaks of opacity, often radiating from the periphery toward the center, which appear black from the interference with the reflection of light from the choroid. In like manner the nucleus may be seen to be hazy and the periphery clear, or the sectors of the lens are strongly marked. The beginning of cataract is also made evident by flaws in the lens, which have been compared to cracks in glass, and are known as "striæ of refraction." If the entire lens is opaque, no portion of the pupillary space exhibits any red reflex from the fundus, although a lens which appears completely cataractous through the undilated pupil may exhibit spots of incomplete opacification in the periphery recognized by the transmitted red glare when the pupil is dilated. The final examination with transmitted light should be made with a +16 D lens or with a corneal loupe.

With *oblique illumination* (see page 51) the opacities, if incipient, appear as white or gray streaks and dots.

When a progressive senile cataract is fully matured, its presence may often be detected without any special examination except in the instances already mentioned, but it is a matter of the utmost importance to ascertain when this full maturity has been reached, or, in other words, whether the cataract is *ripe*. This is determined in the following manner:

The patient being placed in the proper position, the pupillary space is illuminated. If the opacity is complete, the opaque lens, covered by its capsule, is level with the margin of the pupil, and there is no shadow; if not, the major portion of the opacity is at a level posterior to the plane of the pupil, or, in other words, a clear or partly clear space is present between the iris and the opaque portion, and a dark semicircle appears upon the opacity at the side from which the light comes. This is the shadow of the iris. Shining sectors or the transmission of a red glare

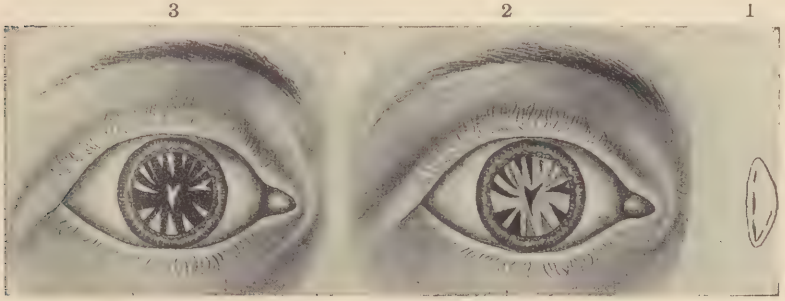


Fig. 183.—Cortical cataract: 1, Section of lens, opacities beneath the capsule; 2, opacities seen by transmitted light (ophthalmoscope mirror); 3, opacities seen by reflected light (oblique illumination) (modified from Nettleship).

indicate immaturity, even if the shadow is absent. In hypermature cataract the shadow is visible, but the surface of the lens is flat.

Development, Course, and Pathology of Cataract.—In progressive *senile* or, as it is sometimes called, *simple* cataract there is a period of growth from incipiency to full maturity which varies considerably, and ordinarily consumes from one to three years. Often the rate of increase is very slow, and immature cataract, especially of the cortical variety, may remain unchanged for many years. At other times the development of the disease is comparatively rapid. This slow progress of cortical senile cataract should be remembered, and the discovery of striæ in the lens need not condemn the patient to rapid deterioration of vision. Indeed, certain lenticular opacities remain practically stationary for years.

The opacities begin either *equatorially—i. e.*, at the edge of the lens—or *centrally—i. e.*, at the nucleus. In the former case the striæ begin just beneath the capsule and are seen both in the anterior and posterior portions. Sometimes broad sector-shaped opacities form, or the

opacities may be narrow, or they may appear as delicate radiating lines. They gradually radiate toward the center (encroach on the pupil space), the nucleus becomes hazy and sclerosed, the cortical layers become opaque, and, finally, the cataract is complete. Cataract, other things being equal, is more prone to begin in the lower part of the lens than elsewhere.

In the second variety the nucleus becomes hazy and the surrounding cloudiness always remains the most opaque portion of the cataract, which gradually spreads to the cortex (Fig. 184). Where diffuse clouding occurs first at the central portion of the lens, that is, in the layers immediately surrounding the nucleus, marked depreciation of vision is an early symptom. A frequent lesion is a disk of opacity in the posterior layers of the cortex, web-like in structure, or sometimes looking like a collection of minute bubbles and striæ. This also causes marked disturbance of vision.

To a ring of opacity near the equator of the lens and behind the iris, seen in old persons and frequently stationary for long periods of time, the name *arcus senilis lentis* has been given.

Fig. 184.—Nuclear cataract: 1, Section of lens, central position of opacity; 2, appearance by transmitted light; 3, appearance by oblique illumination (modified from Nettleship).

Cataract may also begin as a more or less diffuse clouding or in the form of small dots, or blister-like bodies, scattered through the cortex, or located at either pole, or in opacities which, with transmitted light, resemble dark flocculent precipitates. In the last-named circumstances the advance is more rapid than where the striæ are the first manifestation. Instead of going on to maturity, a nuclear haze or a spear of opacity may remain stationary, or at least show no practical change for years.

During the formation of cataract the following changes occur in the lens: First, there is a separation of the lens-fibers with a collection of fluid between them, which coagulates into drops—the spheres of Morgagni. Later there are swelling, clouding, and fatty degeneration of the cortical fibers and the formation in them of nucleated vesicular bodies. Ultimately there is disorganization of the fibers, and the lenticular tissue is changed into fat-drops, spheres of Morgagni, and albuminous liquid, and the cortex separates from the capsule, the

liquor Morgagni collecting between them. The lens nucleus becomes sclerosed, but in other respects is not greatly altered. Paul Römer has studied the pathogenesis of cataract from the point of view of serum investigations, and believes it is possible that, as a result of the degenerative processes of old age, antibodies are liberated in the blood which possess a definite affinity for certain parts of the lens protoplasm. They, by uniting with corresponding receptors of the lens protoplasm, are able to damage the lenticular cells, just as blood-corpuscles are destroyed by the union with them of specific cytotoxins.

Maturity may be succeeded by the stage of "overripeness" and the cataract gradually shrinks to a flat disk, or the liquefaction of the cortical matter permits displacement of the nucleus, a type which is known as *Morgagnian cataract.* Tremulousness of the iris is seen in overripe cataracts. Sometimes calcareous degeneration in the lens or its capsule may take place.

The cataract, the development of which has just been described, is, for the most part, *hard—i. e.*, the nucleus of the lens is large. Under the age of thirty-five all cataracts are *soft—i. e.*, the nuclei are small or wanting, just as the lenses in which they develop have failed to attain the density which later they assume.

Causes of Cataract.—1. *Age of Life.*—Cataract which becomes complete is especially frequent after fifty years; but, as Fuchs remarks, it cannot be regarded as a physiologic attribute of old age. It is a pathologic process, and age, while it is an important factor in its development, must often be regarded only as a predisposing cause. Occasionally total cataract without apparent constitutional disease is found in adolescents. The very beginnings of cataract, according to one observer, are not peculiar to old age, but appear between the twentieth and the thirtieth year as an equatorial cataract.

2. *Sex.*—This appears to have no decided influence, the sexes being about equally affected, unless it be in the zonular variety, in which a greater liability of females has been recorded.

3. *Disease.*—Sugar has been found in the urine of about 1 per cent. of cataract cases, and the cataractous lenses of patients the subjects of diabetes mellitus at times contain sugar. According to Klein, posterior polar, combined with posterior cortical, cataract, unassociated with choroiditis, is significant of diabetes. Albumin is present in about 6 per cent. of the cases, but the etiologic relation of nephritis to cataract has not been proved. Grilli's researches lead him to believe that cataract is caused by a species of dehydration of the lens, brought about by an insufficient elimination of solids by the kidneys, and a consequent rise of osmotic tension in the blood and aqueous humor. Doubtless, changes in the osmotic pressure of the fluids circulating around the lens, whether they are due to toxic causes or local disease, contribute to the formation of cataract, although Römer has shown that they may vary greatly without affecting the lens.

Cataract has also been noted in connection with idiopathic fevers and allied diseases, with gout, malaria, influenza, rachitis, syphilis

(Bos), angiosclerosis, and especially atheroma of the carotid (Michel), epilepsy, and other convulsive seizures, meningitis (Bock), certain cutaneous affections (Mooren, Rothmund, Andogsky—*dermogenetic cataracts*), with bronchocele, with hookworm disease (Calhoun), and sea-sickness (Weeks). As Becker has stated, however, a connecting link between constitutional maladies and opacities of the crystalline lens has not been established. The frequency with which lenticular opacities, either cortical or at the posterior pole, appear in eyes the retinal arteries of which show signs of degeneration (see page 453) is well known. The late D. W. Greene studied the relationship between increased blood-pressure and the formation of cataract, and believed that increased arterial tension exerted a certain influence in the causation of lenticular opacities. It is possible that sclerotic changes in the nutrient vessels of the anterior uveal tract may aid in the development of lenticular degeneration. Schiötz has described a relationship between cataract and disturbances of internal secretion.

4. *Occupation.*—Cataract is especially frequent among glass-blowers, and is attributed to the effect of the radiated heat and excessive perspiration. In *bottle-makers' cataract* the lesions often consist of a dense, well-defined disk of opacity in the center of the posterior cortex, surrounded by smaller opacities. It is not improbable that additional investigations such as the author made years ago would show the same liability to cataract in puddlers and others exposed to intense heat; indeed, Cridland has observed in furnace-workers cataract resembling that which is frequent in glass-blowers, to which the name *furnace-workers' cataract* has been given.

5. *Heredity.*—Remarkable examples of the influence of heredity in the formation of cataract have been published. It has been noted that the tendency is more marked in the child-bearing period, and that the transmission is through the female line; transmission through the male line only, however, has been recorded. All phases of this subject were elaborately studied by the late Mr. Nettleship. In both groups of acquired cataract, that is, senile and presenile or juvenile cataract, the transmission is almost always direct. Occasionally a generation is skipped. Referring to the frequency of *inherited cataract*, he found that women are somewhat more liable to familial acquired cataract than men.

6. *Toxic Agents.*—Cataract has been produced artificially by poisoning rabbits with naphthalin (*naphthalin cataract*), sodium nitrate, and other toxic agents. In addition to the cataract, there are changes in the retina and vitreous and also general disturbances.

During epidemics of *ergotism* patients are at times affected with cataract (*raphanic cataract*), the appearance having been noted almost exclusively in the convulsive type of this toxemia; hence it is not certain whether the lenticular opacity results from the poisoning by the ergot or on account of the convulsions.

7. *Traumatism.*—Cataract may follow a *direct* injury to the lens, or be caused in an *indirect* manner—for example, by a concussion (*con-*

cussion cataract). To this category belong those cataracts which have followed a *lightning-stroke*. A number of examples are recorded, both double and single, partial and complete. In addition to the cataract, optic neuritis, optic atrophy, rupture of the choroid, iritis, iridocyclitis, miosis, mydriasis, and palsy of accommodation have been observed. Cataract may also be caused by a violent *electric shock*.

8. *Diseases of the Eye.*—Cataract may be secondary to numerous acute and chronic affections of the eye—viz., iritis, iridocyclitis, iridochoroiditis, choroiditis, detachment of the retina, glaucoma, and diseases of the cornea, especially sloughing ulcers. The frequent coexistence of disturbance of the choroidal coat and incipient cataract has led to the opinion that while opacity of the lens (so-called senile) is a condition commonly seen in advanced life, it does not, in all probability, depend upon senile changes, but is originated in local pathologic states involving the nutrition of the eye itself (Risley).

9. *Accommodative Strain.*—Investigations show that a large majority of cataractous eyes are hyperopic and astigmatic, and it has been stated that the danger of cataract is increased where the astigmatism is against the rule and remains uncorrected. The evident prophylactic measure is the use of proper glasses.

The etiology of cataract is by no means always clear, and often several factors are necessary to explain it; sometimes no direct cause can be assigned; frequently there are extra-ocular causes and the cataract results from nutritive disturbances.

The following additional facts in regard to the clinical varieties deserve attention:

I. **Senile Cataract** (*Simple Cataract; Gray Cataract*).—This, representing the type of general cataract which develops after the fortieth year of life, is nuclear, cortical, or mixed in its origin. It may not appear before the sixtieth year. Its course from incipiency to full maturity has been described.

The color usually is gray, and the nucleus, which itself does not become cataractous, but is hardened, may be recognized by its yellowish or brownish hue and its waxy appearance.

If the nucleus is small and the surrounding cortex uniformly white, the cataract is comparatively *soft;* if the nucleus is large and the color of the cataract distinctly gray, yellowish, or brownish, it is *hard*.

Instead of a gray or grayish-white color, the cataract may be yellow or amber, or the sclerosis of the nucleus extends to the cortical substance, so that the whole lens is brownish and the pupil black (*black cataract*). Sometimes there is opacity of the hyaloid membrane, which Fink calls *hyaloid cataract*. Occasionally *cholesterin crystals* may be found in cataracts, not only in the senile, but also in the juvenile variety.

Senile cataract generally is bilateral, one eye being more affected than its fellow; but a cataract may attain maturity in one eye before the other lens is affected (*unilateral cataract*).

II. **Juvenile cataract** is a term descriptive of those opacities of the

lens which occur before the fortieth year of life. To such cataracts the name *presenile* is also given (Nettleship).

In forms of cataract developed in early life the evidence of the influence of heredity is often strong.

General cataracts in young persons (*complete cataract of young people*) may arise without known cause or from one or other of the causes already recorded. These are bluish white, often have a sheen like pearl, and are soft.

III. **Congenital Cataract.**—This may appear as a complete or partial opacity of the lens. In the *complete* form the lens usually is white or bluish-white in color, densely opaque, and *soft*. The eye may be otherwise healthy, or there may be changes in the choroid, retina, optic nerve (congenital amblyopia), and sometimes vices of conformation, as coloboma, microphthalmos, and hydrophthalmos. Disturbances of nutrition during intra-uterine life, changes in the choroid, arrest of development, consanguinity of parents, and heredity have been invoked to explain its existence.

Fig. 185.—Zonular cataract (after Spicer).

There are several varieties of *partial* congenital cataract:

(*a*) *Zonular* or *lamellar cataract* appears, as its name implies, in the form of an opaque layer surrounding the clear, but sometimes cloudy, center of the lens, and is the most frequent form of partial congenital cataract. On the surface streaks of opacity are often evident, which may project into the clear cortex and are called "riders." Usually it is double, but may be unilateral, and is either congenital or forms in early infancy. The cataract is stationary in most instances, but occasionally becomes complete.

If the center of the pupil is examined, a reddish point surrounded by a grayish halo will be observed. When the pupil is dilated with atropin and examined with the ophthalmoscopic mirror, the central dark zone will be apparent, surrounded by a reddish circle, due to the reflection from the fundus passing through the peripheral part of the lens, which remains clear. With oblique light the appearances may be as in Fig. 185. A rare type is several zones of opacity separated by zones of transparency. Patients with zonular cataract act like myopes, and the refraction of the eye may be myopic. Macular changes are not infrequent.

The cause of lamellar cataract is not certainly known. In the congenital variety it is probably due to some developmental defect; in the variety arising in early infancy some fault in nutrition has occurred. Most often the subjects are rachitic, and present the teeth and cranial asymmetry peculiar to this affection. Peters considers tetany a more common cause than rachitis, and Hesse and Phelps emphasize the fre-

quent association of zonular cataract and tetany—both are attributed to parathyroid insufficiency. A history of convulsions is common, and dental defects, which are present in the form of lines, furrows, or terraces, may lie transversely across the incisors or canines. Anatomically, lamellar cataract consists of a narrow zone of degenerative change in the lens-fibers, situated between the nuclear and cortical areas (Lawford).

Fig. 186.—Anterior polar cataract (after Nettleship).

Cataract may develop in later life, that is, between the ages of twenty and fifty, in persons who suffer from tetanic spasms. These patients may also suffer from loss of hair, necrosis of the nails, and chronic skin eruption. To such cataracts the name *tetany cataract* has been given, and they are apparently due to a toxin which affects epithelial structure (E. T. Collins).

(*b*) *Central cataract* (*central lental cataract*) consists of a white opacity in the central part of the lens, due probably to faulty development at an early stage of intra-uterine existence. Sometimes vision is surprisingly good; at other times it may be poor, and defects of development in the eye may be present and nystagmus may develop.

(*c*) *Pyramidal Cataract.*—This is also known as *anterior capsular* or *polar cataract*, and consists of a small, well-defined, pyramidal-shaped or circular opacity due to hyperplasia of the capsular epithelium and degeneration of the lens-fibers in that position. It probably arises in consequence of contact of the lens and cornea in fetal life, which causes an arrest of osmosis of nutritional fluid (E. T. Collins). Mules suggested that these cataracts may be cretified remains of the pupillary membrane.

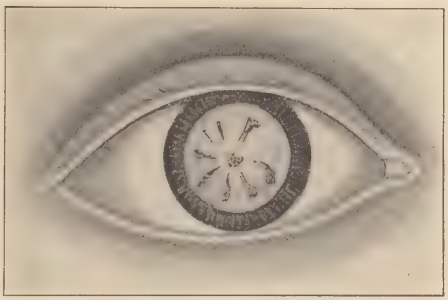


Fig. 187.—Posterior polar cataract seen by transmitted light (from a case of pigmentary degeneration of the retina).

At the posterior pole of the lens an opacity similar to the one described may be found, known as a *posterior polar* or *pyramidal congenital cataract*. It is caused by vestigial remains of the hyaloid artery at its lenticular attachment, or persistence of the part of the posterior vascular sheath of the lens, and, strictly speaking, is not a true cataract, that is, the changes are not in the lens. These opacities are sometimes separated into those which lie beneath the capsule and those which exist upon its surface. A small dot-like opacity of this origin, and which does not disturb vision, is quite common.

(*d*) *Punctate cataract* is an unusual form of congenital lenticular change in which the opacities present themselves in the form of more or less fine points, occupying the center of the pupillary space. The cataract remains stationary for a long time.

(*e*) *Fusiform cataract* is a rare variety characterized by an opaque stripe passing from the anterior to the posterior pole of the lens, sometimes with offshoots, disposed like coral branches. It may be combined with zonular cataract. It is also known as *axial* or *coralliform* cataract, and is prone to occur in families. Nettleship's list contains the record of one family in which thirty members, in four generations, were known to be affected. Nettleship and Ogilvie have described a peculiar form of congenital family cataract in which a disk of opacity, "consisting of a single layer, always thin, but varying in transparency, is situated behind the nucleus, but well in front of the posterior capsule." Disk-shaped opacities behind the nucleus, often hereditary, are also termed "Doyne's cataract."

IV. **Complicated or Secondary Cataract.**—This may be *complete* and arise in consequence of the various diseases of the eye enumerated on page 399. In iritis, for example, fibrinous exudations are attached to the lens-capsule, contraction occurs, the capsule is disturbed in its relation to the underlying lens-fibers, which are separated, and cataract forms. If this process is a limited one, the lenticular opacity may remain circumscribed. It may also be *incomplete*, and appears in the following varieties:

(*a*) *Anterior Polar Cataract.*—In addition to the congenital variety of this opacity there is an acquired type, which arises in consequence of a perforating ulcer of the cornea—for example, in ophthalmia neonatorum (see page 198). In infants' eyes it may follow ulceration of the cornea without perforation.

(*b*) *Posterior polar cataract*, as a congenital variety, has been described; but another form is the more or less star-shaped opacity, which is the commonest variety of complicated cataract, seen at the posterior pole of the lens in high myopia, vitreous disease, disseminated choroiditis, and pigmentary degeneration of the retina. It may remain stationary for a long time, disturbing vision in proportion to its density, or it may progress and become complete. Other forms of posterior polar lenticular opacity, unconnected with evident disease of the ocular coats, are not infrequent. The opacity may be circular, irregularly star shaped, and resemble a small meshwork interspersed with dots and blebs. It may remain unchanged for long periods of time, or the remaining part of the lens may gradually become opaque. A similar appearance toward the anterior pole of the lens is sometimes visible; occasionally the opacity exists in both positions at the same time.

A form of complicated family cataract has been described by Purtscher and by Zentmayer. It occurs about the age of thirty in eyes with thin, bluish-white scleras, tremulous gray-brown irides, deep anterior chambers, contracted pupils, and a tendency to glaucoma after operative interference.

V. **Traumatic Cataract.**—This is caused by *direct* injury to the lens by some penetrating substance which lacerates the capsule and then permits the entrance of the aqueous humor. (See also page 400.) The lens substance swells up, becomes opaque, and some of it may escape into the anterior chamber. Absorption takes place in about six weeks. This course represents the most favorable outcome of such an accident. In other cases there may be iritis, cyclitis, and secondary glaucoma, owing to swelling of the lens.

Instead of going on to complete opacity, an injured lens, in some instances, presents a limited opacity, which remains stationary; in other instances this disappears, and in still others there is slow advance of the opacity.

The opacity was explained by Marcus Gunn by the action of the sodium chlorid of the aqueous humor upon the globulin of the lens substance. This explanation, according to C. A. Clapp, is inconsistent with chemical facts. He believes that when "the lens-fibers are broken up they undergo autolytic changes."

A more *indirect* mechanism of traumatic cataract is *concussion* (*concussion cataract*)—a blow upon the eye causing a slight rupture of the anterior or posterior capsule, followed by opacity, which may become general or retain a limited size for a long time. According to Nettleship, absorption of a complete concussion cataract is more uncommon than where the lenticular opacity has followed a direct trauma, although the lens may gradually shrink in size. A *ring-shaped opacity* may appear on the surface of the lens after contusion of the eye, generally in young eyes. This is a circular opacity, 3 to 4 mm. in diameter, concentric to the margin of the pupil, and of a brownish color. The lesion is sometimes called *Vossius' ring*, or *contusion-lesion of the lens* (Caspar). Vossius ascribes the lesion to pigment adherent to the capsule or to disturbance of the epithelium. It is, as it were, a cast of the iris margin and its pigment on the capsule. In several eyes studied by the author the lesion disappeared within a few weeks after the injury.

VI. **After-cataract.**—This name has been applied to those changes which occur in the capsule of the lens remaining after the extraction of cataract. It is usually called *secondary cataract.*

These changes may depend on proliferation and thickening of the capsular epithelium; on agglutination of the two layers of the capsule, the anterior part being so folded over that it has retained cortical material, which has thus been shut off from the dissolving action of the aqueous and remains as a membranous opacity; or upon new-formed tissue between the capsule layers or thickened elements from the anterior part of the vitreous. If there has been postoperative reaction, fibrinous exudation from the iris adds to the opacity. The name has also been given to the dense white membrane (*membranous cataract*) which is composed of deposits of lymph, and fibrinous and plastic exudation, and to which the iris and even the ciliary processes are adherent, and which follows postoperative iridocyclitis. (Consult Fig. 354.)

VII. **Capsular Cataract.**—The name *capsular cataract* is applied to thickenings and proliferations of the capsular epithelium, and sometimes to subcapsular degeneration of the lens-fibers, which may be congenital, may follow inflammatory processes of the eye (corneal ulcer), and may occur in connection with other degenerations in overripe cataract.

VIII. **Capsulolenticular cataract** is the name applied to opacity of the lens associated with thickening of the surrounding capsule, most commonly in the center of its anterior portion.

Prognosis.—Incipient cataract in the form of striæ in the anterior cortex need not doom the patient to rapid deterioration of sight, because the existing vision is often maintained for long periods of time. *Spontaneous disappearance of senile cataract* has been reported. According to Pyle, this may occur on account of ruptured capsule, dislocation, or degenerative changes; rarely the phenomenon has been observed, although the history of such etiologic relationship could not be obtained.

Operation is generally deferred until the cataract is *ripe*. The surgeon must ascertain whether the eye itself is in a healthy condition by attention to the following considerations:

(*a*) *The probable condition of the interior of the eye*, if no data of ophthalmoscopic examinations during the incipiency of the cataract are at hand. This is ascertained as follows:

Place the patient before a lighted candle about 4 meters distant—the flame should be distinctly recognized. This gives evidence that the macular region is free from extensive disease, but does not exclude a small lesion. Next cause the eye under examination to fix the flame attentively, and move a second lighted candle radially through the field of vision. The flame should be recognized as soon as the rays strike the edge of the cornea, and the patient should be able to indicate the direction in which it is coming. Thus the "light-field," or the "projection of light," is tested, and, if the answers have been accurate, "projection of light is good in all parts of the field."

If the patient fails to appreciate the candle-flame in any portion of the field, coarse changes may be suspected—*e. g.*, extensive choroiditis, detachment of the retina, glaucoma, etc.; but it is not possible to detect a small area of central choroiditis by this means (see also page 353). The macular region should be investigated by requiring the patient to note the separation of two small centrally placed flames, or by causing him to look at the light through a small aperture in the center of a disk. Fluid vitreous, indicated by tremulousness of the iris, is an unfavorable sign. Should there be no light-perception, the cataract is an unsuitable one for operation except that in certain circumstances its extraction is justified in order to improve the patient's appearance.

(*b*) *The Probable Condition of the Refraction.*—It may be impossible to ascertain this unless some record is at hand of an examination when the media were still clear. Some idea of the refraction is obtainable by examining the glasses which the patient may have used during his reading days. High myopia renders the prognosis less favorable; in-

deed, the vision after operation in myopic cases, other things being equal, is not so good as that of hyperopes.

(*c*) *The Mobility of the Iris; Its Reaction to a Mydriatic.*—This should be prompt and normal. Failure of iris reaction in either case may indicate imperfect conductive power in the optic nerve, or atrophy or other change in the iris.

(*d*) *The Age and General Condition of the Patient.*—Advanced age alone does not militate, as much as it would seem likely to do, against successful cataract extraction. So, too, the extraction of diabetic cataract is often followed by good results; and even the presence of chronic Bright's disease, while a complicating circumstance, does not forbid the operation. Great feebleness, dementia likely to become worse with confinement, nasopharyngitis, advanced arteriosclerosis, eczema, cystitis, and chronic bronchitis are unfavorable conditions. According to Hansell, syphilis should be regarded as a dangerous complication.

(*e*) *The Condition of the Area of Future Operation and of Its Surroundings.*—Disease of the lacrimonasal channels, trachoma, chronic conjunctivitis, and blepharitis contraindicate cataract extraction because the wound is almost certain to become infected by the unhealthy discharges. In such circumstances a line of treatment later described must be instituted before operation. A matter of importance, not always attended to, is the state of the rhinopharynx. This should be reasonably healthy to secure the highest type of success. Eczema of the face or other regions of the body is a source of danger. Prior to cataract extraction a careful bacteriologic examination of the conjunctiva is important.

(*f*) *The Type and Condition of the Cataract.*—In making a prognosis the size of the nucleus and its position, the probable consistence of the cortex, the primary or secondary nature of the cataract, and its stage of maturity must be considered. Certain conditions (amblyopia) influence the prognosis in complete congenital cataract, and in the partial varieties, like the lamellar form, the eye may be defective in construction. In traumatic cataract the extent of injury to parts other than the lens must be regarded.

Treatment.—This may be divided into the treatment of *immature* and of *mature* cataract.

Drugs do not exist which can dissolve a growing cataract; and the use of electricity, which has been recommended, is of no value. Massage of the eyeball associated with the instillation of a mixture of glycerin and boric acid solution has been commended (Kalish). The author has failed to observe the slightest benefit from such treatment.

1. The refraction should be carefully tested and that glass ordered which gives the most accurate vision. It may be necessary to make frequent changes in the correcting lenses to conform with the alterations in refraction brought about by the swelling of the lens.

2. Congestion of the choroidal coat may be relieved by the exhibition of certain alteratives, among which the iodids of sodium and potassium

and syrup of hydriodic acid are the most suitable. The iodids may be combined with small doses of bromid of potassium or bromid of sodium. Subconjunctival injections of iodid of potassium have been recommended (Badal, von Pflugh); of their value the author has no knowledge. Dor advocates the treatment of cataract locally with a solution composed of 5 grams of desiccated sodium iodid, 5 grams of crystallized calcium chlorid, dissolved in 400 grams of distilled water. The solution should be applied for half an hour a day by means of a glass eye-bath. The daily instillation of a 1 per cent. solution of iodid of sodium or iodid of potassium has also been tried. Some observers report favorable results from the persistent use of dionin in solutions of gradually ascending strength. Col. Henry Smith strongly recommends subconjunctival injections of cyanid of mercury (1 : 4000–6000). The moderate use of the eyes may be permitted without danger of hastening the process of maturation. If the patient suffers from diabetes, nephritis, lithemia, or arteriosclerosis, suitable dietetic and medicinal measures should be employed. Drinking water freely is advised by Edward Jackson.

3. Often comfort may be given and vision improved by keeping the pupil dilated with a weak mydriatic (if the opacity is central). Tinted lenses, which correct any existing refractive error, should be worn. In other cases a miotic is useful.

If the vision of eyes suffering from cataract of the nuclear type is improved by mydriasis, this has been given as an indication for *optical iridectomy*, but is not a sufficient one unless the patient finds by observation that the increased visual acuteness, as noted by test-type examination, is also advantageous in pursuing his ordinary occupation.

Artificial Ripening.—The exceeding slowness with which a senile cataract may progress often leaves the patient in a state of semiblindness. To remedy this, several methods have been proposed for hastening the process of ripening.

Simple division of the anterior capsule; division combined with iridectomy (Mooren); division and external massage (Rohmer); iridectomy and triturating the lens-fibers by rubbing the cornea over the coloboma with a horn spoon (Förster's method); paracentesis of the cornea, and internal massage directly on the anterior capsule with a small spatula (Sasso and Ricaldi and B. Bettmann, of Chicago); and simple paracentesis of the cornea with external massage (T. R. Pooley, of New York, and J. A. White, of Richmond, an operation practised by the latter surgeon with much success).

A discission, after the manner of Graefe, carried deep into the lens substance, was recommended by Schweigger as the only satisfactory method, especially before the fortieth year; and a *preliminary capsulotomy* has been advised and practised by Homer E. Smith (see page 669). He has reported excellent results, as have other surgeons who have followed his method.

Treatment of Immature Cataract.—Some operators of extensive experience hold that the usual criteria of ripeness are erroneous in that period when accommodation is annulled by physiologic changes in the

lens—that is, about the sixtieth year—and the lens may be extracted safely even if it is in part unclouded. It may also be done successfully at an earlier age.

Many operators, following McKeown's advice, after the extraction of immature cataract resort to intracapsular irrigation and wash out tenacious cortical material with a suitably warmed physiologic salt solution. Indeed, irrigation of the anterior chamber forms part of the technic of all cataract operations in the hands of some surgeons. Suitable irrigation apparatus has been designed by McKeown and by J. A. Lippincott.

If the unripe material is not removed it may swell up and cause iritis. Therefore the safest plan is to wait for maturity; but if this is impossible or very undesirable, the author has been in the habit of extracting an unripe cataract in preference to performing a ripening operation. With irrigation of the anterior chamber the author's experience has been comparatively limited, but the testimony, constantly increasing, of its value in removing cortical material and aiding in a restoration of tone to the eye tissues, especially the iris, is so clear that evidently it represents a procedure which should be considered in all suitable cases.

According to Colonel Henry Smith, Derrick Vail, W. A. Fisher, and a few other surgeons, the best means of managing immature cataract is its *extraction in the capsule* by the so-called Indian or Smith method (see page 660). The stage of immaturity at which Smith advocates the extraction of immature cataract in the capsule is where the opacity has progressed so far as to unfit the patient for the performance of his ordinary duties. With this operation the author has had no experience. If the patient is unable to wait for maturity of the cataract, he has often successfully extracted an unripe lens after preliminary iridectomy, and recently has employed irrigation with normal saline solution to get rid of cortical remnants. In a few instances he has practised deep discission or preliminary capsulotomy.

Treatment of Mature and Complete Cataract.—*Mature* cataract requires an operation for its removal, differing according to the age of the patient and the consistency of the cataract.

Hard cataracts, or those which occur after the fortieth year, are suitably removed either by—(*a*) simple extraction (extraction without iridectomy), (*b*) combined extraction (extraction with iridectomy), or extraction after preliminary iridectomy.

In recent times extraction in the capsule, according to the Smith method, has been extensively tried.

Soft cataracts, or those which occur before the thirtieth year, are suitably removed by—(*a*) linear extraction; (*b*) the needle operation, or that of solution by discission; and (*c*) the suction method. A soft cataract before the twenty-fifth year may be removed through a linear incision into the cornea, and a semifluid one by suction. Complete cataract of young people and complete congenital cataract are generally removed by discission, the latter variety of cataract being ready for operation after the completion of dentition.

According to E. Treacher Collins, a child should be ten months old before operating for congenital cataract. If the pupil is small and does not dilate with atropin, an iridectomy may first be necessary. In so-called *disk-shaped cataract*, that is, where an anterior polar one is set in a ring of clear or partially clear lens substance, an attempt should be made to dislocate it with a needle and let it fall into the anterior chamber.

Treatment of Partial Congenital Cataracts.—Central, lental, and zonular cataracts are treated by iridectomy or by discission. The former procedure is better if, after dilatation with a mydriatic, there is sufficient improvement in vision to justify the manufacture of a new pupil or glasses do not improve vision. This should be made opposite to the clearest part of the lens. If this does not prove satisfactory, the lens may be needled or, finally, the entire lens may be extracted.

Pyramidal, punctate, and fusiform cataracts are not generally amenable to operative treatment. Discission or one of its substitutes is the method of operating applied to after-cataracts.

Extraction of *unilateral cataract* will not usually give the patient increased visual acuteness because, owing to the inequality of refraction, the eyes will not work together. The operation may be performed (simple extraction) for cosmetic reasons, to avoid overmaturity in the opaque lens, and to improve the field of vision upon the affected side. If there is divergence, a subsequent tenotomy of the externus or advancement of the internus may be necessary.

The technic of performing the various methods of cataract extraction, the dangers and accidents, will be described on pages 647–669.

After a successful extraction or solution, and after sufficient time has elapsed to secure firm healing, a suitable pair of lenses should be adjusted—one for distant vision and one for reading.

Removal of the crystalline lens produces the condition technically spoken of as *aphakia*, and causes a high degree of hyperopia, in the emmetropic eye corresponding to about 11 D. The degree of hyperopia will be diminished if the previous refraction has been myopic, and it is possible to produce emmetropia provided the former near-sightedness has been of such degree that the removal of the lens exactly neutralizes it (see also page 136).

In ordinary circumstances the correcting lens for distant vision is about +10 D. For reading and similar occupation a lens having a focal distance of 25 to 33 cm. is added to the distance glass.

In addition to the hyperopic refraction which follows cataract extraction, a certain amount of regular astigmatism is the result of the operation, due probably to failure of the wound to heal evenly on account of inaccurate coaptation of its edges, caused by the character of the incision or by some condition—for example, badly applied dressings—during convalescence. This astigmatism is generally "contrary to the rule," and is often higher during the first month or two after the extraction, or until cicatrization is complete, than it is at a later period. Usually not more than 3 D remain permanently, but even 1 D should

be sought out and corrected. Naturally, prolapse or incarceration of the iris causes a very high degree of astigmatism.

Glasses should not be adjusted until all redness has disappeared from the eye, and they should not be worn constantly at first. It is wise to wait from six weeks to two months before ordering the glasses for constant use.

The amount of vision obtained after a cataract extraction varies quite considerably. Perfect vision is frequently secured—*i. e.*, $\frac{6}{6}\left(\frac{20}{xx}\right)$, but more often patients must be content with lower degrees, $\frac{1}{8}$ or, according to some operators, $\frac{1}{10}$ of normal vision being considered sufficient to place the case within the category of successes.

Acuteness of vision may be considerably raised by successfully dividing the capsule of the lens which remains behind, and some surgeons perform this operation almost as the rule (see Operations).

Fig. 188.—Spontaneous dislocation of lens into the anterior chamber of highly myopic eye (from a patient in the Philadelphia General Hospital. Drawing by Dr. Randall).

Dislocation of the Crystalline Lens.—This may be congenital (*ectopia lentis*), and is due to a relaxation or absence of the zonula. The displacement ordinarily is *incomplete*, and really consists in a decentration of the lens; but *complete* congenital luxation is also described. Congenital cases are usually symmetric, and generally the displacement is lateral, upward, or upward and outward. But in the course of time the lens may leave this position, owing to elongation of the zonular fibers, and be displaced downward and outward. Several members of the same family may be affected; for example, G. Griffin Lewis has reported *hereditary ectopia lentis* extending through six generations and involving sixteen individuals. Unilateral cases are also described.

In addition to congenital dislocations, there are those due to disease of the eye—*e. g.*, choroiditis, malignant myopia, etc.—and those caused by traumatism. Traumatic dislocation may also be *incomplete* or *complete;* if the latter, the lens may be dislodged from its normal position

backward into the vitreous, forward into the anterior chamber, or, through a wound, beneath the conjunctiva, and even under Tenon's capsule.

Symptoms.—If the dislocation is partial, the margin of the lens may be seen as a dark line with the ophthalmoscope, the refraction of the eye will vary according to the point through which it is observed (*i. e.*, through the lens or beyond it), the iris is tremulous from loosening

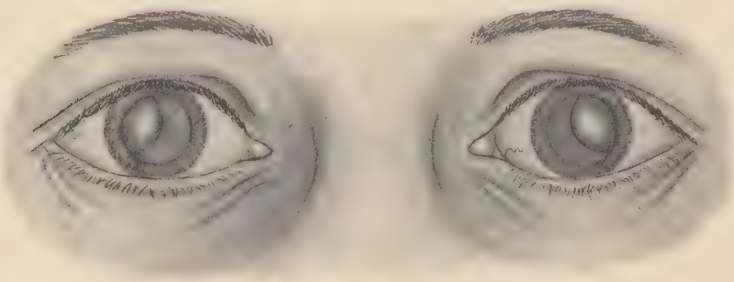


Fig. 189.—Congenital dislocation of the crystalline lenses, up and out (patient in the University Hospital).

of the suspensory ligament and lack of the support of the lens (*iridodonesis*), and monocular diplopia and impaired or absent power of accommodation are demonstrable. If there is complete posterior luxation, the symptoms are much the same as when the lens has been removed by operation, and if the cause of the dislocation is trauma, the symptoms of the injury—*e. g.*, hemorrhage, etc.—may be present.

Fig. 190.—Subconjunctival dislocation of the lens (from a patient in the Chester County Hospital).

A dislocated lens usually becomes cataractous, and often causes intense pain and frequent attacks of iritis, or, by occluding the angle of the anterior chamber, may give rise to glaucoma.

Treatment.—In partial dislocation an attempt should be made to secure the best vision with suitable glasses. Sometimes it is possible to remove an incompletely congenitally luxated lens by linear extraction following a discission.

In complete luxation into the anterior chamber the lens may be removed by a simple corneal incision. For removal of a lens dislocated into the vitreous humor, provided it is producing irritation, a scoop introduced through a peripheral corneal incision may be employed, or the operation devised by the late C. R. Agnew may be attempted. In the latter, a double needle or "bident" is thrust into the vitreous humor far enough back to avoid wounding the iris, the handle of the instrument is depressed, the lens is caught and brought forward through the pupil into the anterior chamber, and removed in the ordinary way. Knapp prefers, in these circumstances, after thorough local anesthesia, to expel the lens by methodical external pressure through an upper corneal section, after removal of the speculum. He presses the edge of the under lid on the lower part of the sclera, directly toward the center of the eyeball. If this fails, he introduces a wire or metal spoon through the corneal section and the pupil, and extracts the lens in this way. The author has employed this method with satisfaction.

If the lens has been dislocated beneath the conjunctiva, it should be extracted through a small incision made directly over it.

After the successful removal of a dislocated lens the eye should be provided with cataract glasses.

Foreign Bodies in the Lens.—Foreign bodies lodged in the lens usually cause general opacity. Occasionally the body is surrounded by a small opacity which remains localized, the remainder of the lens being clear. If a piece of steel or iron is embedded in the superficial layers, it may be dislodged with the electromagnet, and even from the deeper layers by the powerful magnet of Haab. If the lens is opaque the whole crystalline lens, with the foreign body in it, should be extracted, lest the foreign body become displaced and disappear within the eye. If any difficulty is experienced in deciding the position of the foreign body, or whether a foreign body is really in an opaque lens, the Röntgen rays should be employed. A properly prepared series of skiagrams will practically always decide the question.

CHAPTER XIV

DISEASES OF THE VITREOUS

Inasmuch as the vitreous after birth contains no blood-vessels and is not subject to inflammation, it has been contended that the term *hyalitis* is not correct. Straub, however, defends its use in that he believes that irritants in the vitreous may produce chemotactic substances which attract leukocytes from the vessels of neighboring structures, and the process should be regarded as a true inflammation.

Pus in the Vitreous (*Abscess of the Vitreous*).—This condition is caused by a penetrating injury, a foreign body, or a purulent choroiditis, for instance, a metastatic choroiditis after inflammation of the cord in newborn children, or after scarlet fever, erysipelas, relapsing fever, basic meningitis, cerebrospinal meningitis, etc. (see also page 358).

Purulent collections in the vitreous may complicate the infectious diseases or may be caused by an infection which passes through an operation scar from a few months to many years after apparent healing. Cystoid cicatrices are particularly dangerous in this respect. The entrance of bacteria may be facilitated by the presence of prolapsed iris tissue, thinness of the scar, a fistula, and insufficient nourishment of the cicatricial tissue. On experimental grounds a defect in Descemet's membrane appears to be necessary to permit the microbes to pass into the deeper tissue of the eye. Even when the scar is dense and there is no fistula, bacteria may enter (Dolganoff and Sokoloff).

Symptoms.—If the cornea is clear, a yellowish reflex is seen shining through the pupillary space, there are retraction of the periphery of the iris and bulging of its pupillary border. Usually one or two synechiæ are present and the tension is diminished. In addition to this, there may be a pericorneal zone of congestion connected with the inflammation of the iris and ciliary body.

If the exudation in the vitreous is circumscribed, the symptoms at the first glance are not unlike those of glioma of the retina, and the name *pseudoglioma* has been given to this condition, especially as it is seen in children. It is, however, to be distinguished from a true glioma of the retina by the history of the case, the usual presence of the signs of iritis, the retraction of the periphery and bulging of the pupillary border of the iris, and the diminished tension of the globe.

These cases of *pseudoglioma* or *ophthalmitis* are especially noteworthy as they occur in children and young subjects suffering from meningitis. There is purulent inflammation of the uveal tract, with deposits of exudation in the vitreous which give rise to the yellowish appearance which can be seen through the pupil. The retina is detached and the optic nerve inflamed. The affection has been attributed to an extension of inflammation from the meninges along the optic nerve, but

Percy Flemming suggests that the meningitis and ophthalmitis is each the result of a pyemic process. The source of the pyemia may be middle-ear disease. Stephenson urges examination of the pus in the eye for the meningococcus (Diplococcus intracellularis meningitidis). This micro-organism is also responsible for some cases of purulent conjunctivitis (see also page 189). Among 43 cases of ophthalmitis there have been 7 deaths, 6 from meningitis (see also page 357). George Coats and J. Graham Forbes have carefully investigated the relation of the *Meningococcus intracellularis* to pseudoglioma, and believe that a causal relation may be established between this organism and this disease.

Treatment.—If pus has once formed in the vitreous, in the manner just described, no medicinal treatment is of avail; the ball will shrink and enucleation is usually necessary. Intra-ocular injections of chlorin water have been recommended.

If, during the earlier stages of this affection—for instance, during the course of a low fever—the discovery is made that fine flakes of opacity are beginning to appear in the vitreous, it is possible that a vigorous supporting treatment may save the eye from destruction (Hansell).

Opacities in the Vitreous.—These are either *fixed* or *moving*, and vary considerably in shape, size, and somewhat in color. The opacities may appear in the form of membranes, bands, dots, threads, flakes, and strings; or, finally, the entire vitreous humor may give evidence of uniform loss of translucency, which on careful focusing resolves itself into a diffuse, dust-like opacity.

The fixed membranous opacities usually are adherent by two or more points to the choroid, retina, optic disk, and sometimes to the ciliary processes, and even to the posterior capsule of the lens. They may exist as a membrane which crosses the vitreous and covers the optic disk, or as membranous bands running from before backward, and may be coarse, dense, and organized, or fine and more like a cobweb in texture.

Method of Detection.—The examination of the vitreous is made after the manner described on page 99.

The rapidity with which the bodies move depends upon the consistency of the vitreous humor; if this is normal, the movement is slow; if it is fluid or semifluid, the movement is correspondingly rapid.

The different layers of the vitreous may also be examined for fixed opacities by means of the upright image in the ordinary way, by first finding the optic papilla, then gradually placing stronger and stronger convex lenses behind the sight-hole of the mirror until a + 16 D is in place, thus bringing everything into focus from behind forward. The observer's head must be close to the observed eye.

The *subjective* symptoms of vitreous opacities depend entirely upon their amount and density. There may be little or no depreciation of central vision, or this may be diminished and even entirely obliterated. Patients frequently are conscious of black and gray spots before their eyes; sometimes these assume fantastic shapes, and not infrequently these shapes repeat themselves so constantly that the patient is able accurately to describe them. The same symptoms may appear where

there is no organic disease (see page 417). Alterations in the field of vision, pain, redness of the eye, or similar conditions will depend largely upon associated changes, and usually are absent if the vitreous alone is affected.

Causes.—1. *Refractive error,* generally high degrees of myopia associated with changes in the choroid and posterior staphyloma.

2. *Diseases of the eye,* chiefly cyclitis, iridocyclitis, uveitis, choroiditis, and retinitis.

The shape and character of the opacities vary with the condition which has caused them. In cyclitis and iridocyclitis inflammatory opacities are seen; in chronic and old-standing choroiditis flake-like or thread-like opacities are very common, especially in elderly people, and are probably due to hemorrhages having their origin in the choroid. In syphilitic choroiditis and retinitis, in addition to large, floating opacities, there may be a diffuse mist which resolves itself into the so-called *dust-like opacities* (*hyalitis punctata*), and is almost characteristic of the disease which has caused the original inflammation of the choroid and retina. These dust-like opacities are either diffused throughout the entire vitreous chamber, or are situated in its posterior layers, or anteriorly, in the neighborhood of the ciliary region.

3. *Injuries of the eye,* which have caused a hemorrhage from the choroid or ciliary region. The origin of the opacity is an extravasation of blood. In the latter case, as has already been mentioned, suppuration of the vitreous may occur.

4. *Diseased Conditions of the System, Local or General.*—Infectious diseases, wide-spread endarteritis; arteriosclerosis, gout, syphilis, tuberculosis, malaria, portal congestion, anemia, and irregular or suppressed menstruation may be responsible for vitreous opacities; also the prolonged action of arsenic.

5. *Absence of Apparent Cause.*—Opacities of various shapes, often fine and thread-like, and commonly seen in old people, occur without evident disease of the uveal tract, retina, or optic nerve. Their presence in some instances is without serious import. Sometimes the vitreous is studded with minute light-colored spheres; probably a congenital condition, named *asteroid hyalitis* by Benson. White, glistening spots in the vitreous have also been described as evidences of *fatty degeneration.*

Prognosis.—This depends entirely upon the cause of the vitreous disease. If this has started in a purulent disease of the choroid or a purulent change in the vitreous has taken place, the prognosis is exceedingly unfavorable, and the eye goes on to destruction.

If the cause of the disease is syphilis or other constitutional condition amenable to treatment, satisfactory clearing of the vitreous may be expected; even very dense opacities will disappear under proper treatment. When the opacities are due to hemorrhage, the absorption of the clot is not so likely to take place. Both hemorrhagic opacities and others are subject to relapses.

Treatment.—In any case of vitreous opacity, provided the general fundus of the eye-ground justifies this, and there is reason to believe that

eye-strain in any sense is connected with its cause, suitable lenses should be ordered, but the use of the eyes at close ranges should be discouraged.

In syphilitic vitreous disease the usual remedies are applicable. If the vitreous change depends upon an exhausted condition of the system, supportive measures are indicated.

If the patient's condition warrants it, excellent results follow sweats induced with pilocarpin, or by means of Turkish baths or in an ordinary electric lighted cabinet. Iodid of potassium and sodium are useful, as is syrup of hydriodic acid.

If the disease which implicates the vitreous depends upon constipation and portal congestion, in addition to a regulated diet cholagogue laxatives should be administered. Anemia and menstrual irregularities are evident indications for treatment; in the former case the combination of bichlorid of mercury with iron is useful. If there is an active inflammatory condition, blood-letting from the temple may be practised; in fact, the treatment becomes that which is suited to the acute inflammation which has started the disorder. The use of the galvanic current has been warmly recommended by some surgeons in vitreous opacities. Elschnig has treated a certain number of eyes with vitreous opacities following hemorrhage by aspirating ½ c.cm. of vitreous and replacing it with normal saline solution.

Muscæ volitantes (*myodesopia*) are the black specks and motes often seen in the field of vision, especially if the eye is directed toward a bright surface. They follow the movements of the eye, and are especially annoying during the act of reading, as they float across the page. They do not actually interfere with vision.

There is no true opacity of the vitreous, and the ophthalmoscope fails to detect in these instances opaque particles. They are probably due to the shadows thrown upon the retina by naturally formed elements in the vitreous bodies, perhaps the remains of embryonic tissue. Corpuscles in the retinal vessels may be seen by looking through a dark-blue glass at a white cloud. They appear as small oval bodies.

Although of no serious import, so far as sight is concerned, these muscæ produce an amazing amount of annoyance in nervous and sensitive persons. Patients frequently maintain that they obscure an object, floating directly in front of it, and they assume exaggerated and fantastic shapes. They are often ascribed by the laity to disorders of digestion and torpidity of the liver, and are aggravated by the habit which their possessors form of searching for them.

Treatment.—Eye-strain should be removed by the adjustment of suitable lenses, and a course of alterative tonics may be ordered.

Hemorrhage into the Vitreous.—As has already been stated, many vitreous opacities result from hemorrhages from the vessels of the choroid, ciliary body, or retina. Hemorrhage into the vitreous may result from anemia, nephritis, diabetes, arteriosclerosis, myopia, and glaucoma. According to Ridley, if the hemorrhage arises from the retinal vessels, the hyaloid is usually detached and the blood lies between this membrane and the vitreous. If the ciliary body is the source of the

hemorrhage, it usually bursts through the hyaloid into the vitreous. Retinal detachment may occur, especially if the hemorrhage recurs on several occasions. Injury is a common cause of hemorrhage in the vitreous, and in such circumstances the entire chamber may be so filled with blood that it is easily detected in its natural color as a dark-red clot, sometimes being so dense that no reflex comes from the fundus.

Finally, in certain cases, generally in young male adults, *spontaneous hemorrhage* into the vitreous occurs, together with hemorrhage in the retina. According to Eales, such patients are liable to constipation, irregularity of the circulation, and epistaxis. Hutchinson thinks that gout may be a cause in some cases. The investigations of Axenfeld and Stock indicate that an important etiologic factor in recurring intraocular hemorrhage in adolescence, as well as in proliferating retinal lesions and retinal periphlebitis (see also page 440), is tuberculosis, the active agent being a tuberculous toxin. The author has made some clinical examinations in confirmation of this statement. There is marked disturbances of vision depending on the density of the clot, which is likely to be imperfectly absorbed, and both in this form of vitreous hemorrhage as well as in others there may be repeated blood extravasation. To these frequently *recurring hemorrhages into the vitreous* the term "malignant" has been applied. It is possible, as Coats suggests, that anomalies in the coagulability of the blood may account for many of these cases.

Treatment.—This should consist in the administration of cardiac sedatives, laxatives, and later of iodid of potassium or sodium, according to the circumstances. If arteriosclerosis is present, the usual treatment of this condition is indicated. Sometimes instead of elevated there is lowered arterial tension. The administration of calcium salts to aid the coagulability of the blood is worthy of trial; coagulose and pituitrin may be administered and thyroid extract has been successfully employed. Injections of *hemolytic serum* have been tried, but with disastrous results (Elschnig). Intravenous injections of human blood-serum have been used (A. E. Davis). Ligation of the carotid for recurring hemorrhage into the vitreous has been performed, and in a few instances with success. As in other vitreous changes, if the general condition permits it, a sweat-cure may be instituted, either by means of the Turkish bath or with pilocarpin. Enterogenous auto-intoxication should be corrected. Because of the relation of tuberculosis to this condition the advantages of tuberculin treatment should be considered.

Synchysis (*Fluidity of the Vitreous*).—This is a softened or fluid condition of the vitreous, which, as has already been implied, can be positively diagnosticated or, rather, assumed to be present only by noticing the rapid movement of particles of opacity contained within it during motions of the eye. Although tremulousness of the iris is sometimes seen where there is decided fluidity of the vitreous humor, this symptom does not prove its condition, but only a lack of support by the crystalline lens owing to relaxation of the zonula. The tension of the eyeball may be diminished.

It occurs in elderly people with disease of the choroidal coat and in high myopia. A fluid vitreous is a complication of serious import in cataract extraction.

Synchysis scintillans is a term applied to a fluid vitreous which holds in suspension numerous scales of cholesterin which move with great rapidity across the ophthalmoscopic field and produce a striking picture, resembling a shower of brilliant crystals. Poncet has reported in this connection tyrosin and crystallized phosphates, but investigations seem to show that the appearance is due solely to cholesterin.

The affection probably depends upon a choroiditis, and is said to be more common among alcoholic subjects and those with arthritic tendency or any serious disorder of nutrition. The affection is, however, clinically at least, seen in eyes which apparently are not diseased in

Fig. 191.—New blood-vessel formation in the vitreous.

other portions, especially in old people, and may be present in advanced degree without depreciation of visual acuteness.

Treatment.—This does not appear to have any influence. Succinate of iron has been recommended.

Blood-vessel Formation in the Vitreous.—Occasionally cases are examined which present an entirely new blood-vessel formation in the vitreous in front of the entrance of the optic nerve (Fig. 191).

Only a few vessels may be present or, in extreme cases, the entire disk is obscured by a congeries of contorted vessels, the whole forming an extensive vascular veil of anastomosing capillaries coming directly from the nerve-head and having no connection with the retinal vessels (Harlan). The vessels may owe their origin to vitreous and to retinal hemorrhages; in other cases the origin is probably of a specific nature (Hirschberg). The relation of tuberculosis to this and similar conditions has been described.

Foreign Bodies in the Vitreous.—These are usually chips of steel, splinters of glass, particles of gun-cap, or small shot. They may reach the vitreous by penetrating the sclera directly or by passing through the cornea and lens. The foreign body, if unremoved, may cause suppuration in the injured eye or sympathetic ophthalmitis in the fellow eye. The symptoms, diagnosis, and treatment of foreign bodies in the vitreous have been included with injuries of the sclera on pages 293–297.

Entozoa in the Vitreous.—*Cysticerci* in the vitreous are exceedingly rare except in Northern Germany, and here also, according to Hirschberg, their frequency in recent years has much diminished.

Another parasite which has been seen in the vitreous, the removal of at least one specimen being on record, is the *Filaria sanguinis hominis.*

Detachment of the vitreous is a condition which, of itself, would not create blindness; but because it produces detachment of the retina, it is a change of the gravest import.

Traumatism, choroiditis, hemorrhages, intra-ocular growths, and staphyloma may cause it. The vitreous humor is said to be occasionally detached without change in its translucency, although opacities are usually present. Shrinking of the vitreous after a blow on the eye causes its hyaloid to be detached from the retina. In eyes removed after injury, stretching across the globe behind the lens, the so-called *cyclitic membrane* may be seen.

Persistent Hyaloid Artery.—During fetal life the vitreous humor is traversed by the *hyaloid artery,* which is an extension of the central artery of the retina, and proceeds from the optic nerve to the posterior surface of the lens. The vessel passes through a channel having a delicate membranous lining, known as the *canal of Cloquet.* Obliteration of this artery begins at the end of the fifth month of gestation.

Sometimes obliteration fails, and the most important congenital anomaly of the vitreous is evident—namely, the persistence of some vestige of the hyaloid artery. It may appear in the following forms:

A rudimentary strand attached to the disk; a strand attached to the disk and a vestige also at the posterior surface of the lens; a strand passing from the disk to the lens; a similar strand containing blood; a strand attached to the lens alone; and a persistent canal (canal of Cloquet) without any remnant of the vessel. These are the most ordinary and well-recognized forms.

In addition to this, shreds of tissue and membranes on the optic disk, masses resembling connective tissue, and small cystic bodies are probably remnants of this artery. Its rôle in producing posterior capsular cataract has already been described. The appearances are readily recognized by the ophthalmoscope, and require no further description than the names already given.

This classification has been condensed from the admirable monograph of Dr. De Beck who has written a complete account of the anomaly. According to Uribe Troncoso, a free cyst developed from the ciliary processes may give rise to the appearance of a vesicle floating in the vitreous.

CHAPTER XV

DISEASES OF THE RETINA

Hyperemia of the Retina.—Although the capillary network of the retina, invisible in ordinary circumstances, may, under other conditions, become evident (*capillary congestion*), the presence of a congestion is inferred, not by any alteration in the appearance of the retina itself, but by changes in the surface of the optic disk, generally known by the terms *increased redness* or *undue capillarity*, and is associated with increase in the amount of the retinal striation which surrounds the papilla, so that its edges are veiled or slightly blurred. Such appearances are common in asthenopic and ametropic eyes, and in persons whose occupations expose them to the glare of artificial heat—*e. g.*, puddlers.

It is possible to speak with more confidence of a change in the caliber, course, color, and general size of the retinal vessels, provided more than the normal amount of blood finds its way into them and they are distended, tortuous, or positively lengthened. It is customary to describe the hyperemia as *active* if an increased amount of blood is sent to the retina, because the systemic circulation is unduly filled—*e. g.*, in rapid action of the heart with fever, pneumonia, etc.—and as *passive* if the blood is not properly returned from the eye, for example, in compression of the retinal vein. In the last-named circumstance the veins are large, filled with dark blood, and often tortuous, while the arteries are unaffected or are smaller than usual.

Among the *general causes* of a stasis-hyperemia may be mentioned mitral disease, emphysema, violent cough, convulsive seizures, or, in short, any cause which is likely to produce engorgement of the veins of the head and neck, and to prevent the emptying of their contents into the great venous channels of the chest. Increase in the diameter of the veins is much more frequent than increase in the diameter of the arteries, while, on the other hand, increase in the diameter of the arteries is uncommon as compared with a diminution of their caliber (Loring). Pathologic significance must not always be ascribed to apparent changes in the diameter of the veins, because eye-grounds are often crossed by large dark veins, the arteries being small by contrast, without definite local or general cause for the phenomenon.

Ordinarily patients with hyperemia of the retina do not present characteristic symptoms, but if the condition is connected with ametropia there are ocular pain, photophobia, and lack of eye endurance.

Treatment.—In hyperemia dependent upon errors of refraction the evident treatment is physiologic rest under the influence of atropin,

and later a suitable correction with glasses. When the condition depends upon general causes, these furnish the indications for treatment.

Anemia of the retina should be looked upon not as a disorder of this structure, but as a symptom of local pressure or of some cause situated within the general economy.

The highest type of anemia of the retinal vessels is seen with stoppage of the circulation by an embolus or thrombus, and occurs in marked degree as the result of compression, in consecutive atrophy of the optic nerve. Other causes of anemia of the retina are general anemia, cerebral anemia, and syncope.

Extreme narrowing of the retinal arteries is occasionally seen as the result of a vasomotor spasm—for example, in "sick headaches" and in true migraine. In these cases there may be temporary complete or partial (hemianopic) blindness. If the blindness approaches from above downward, the obstruction is in the retinal circulation, but if it assumes a lateral form, the cortical visual centers are probably affected (Priestley Smith). Impeded retinal circulation may be attributed to the high arterial tension which is known to be present in some cases of migraine.

Under the name *ischemia of the retina* a condition is described in which, with complete blindness, there are pallor of the optic disks and extreme narrowing of the retinal blood-vessels. This has been seen in the collapse stage of cholera (Graefe), in whooping-cough (Knapp, Noyes), in erysipelas (Ayres), and under the influence of toxic doses of quinin and salicylic acid.

Treatment.—The flagging circulation should be stimulated by digitalis and strychnin. Nitrite of amyl has been employed in spasm of the retinal arteries. General anemia calls for its appropriate remedies.

Hyperesthesia of the Retina.—This is characterized chiefly by the *symptoms* which indicate a supersensitive state of the retina—dread of light, lacrimation, blepharospasm, neuralgic pain, and imperfect eye endurance.

Ophthalmoscopic changes may be practically absent, but in most instances those lesions will be detected which have been referred to under *congestion,* but which, adopting a name which was originally employed by Jaeger and later used by Loring, may be described as *irritation of the retina.* These are: undue redness of the nerve-head, veiling of its nasal edges, from which, and from those above and below, distinct striation of the retinal fibers are evident, while streaks of light tissue can be followed along the course of the larger vessels. The margins of the disk are veiled by this retinal striation, and although the physiologic cup, or the "light spot," may be unchanged, the general surface of the disk seems to be covered with a delicate layer of edematous tissue. At the same time the choroid reveals changes similar to those described on page 344, or else is distinctly granular and macerated. Often the entire fundus fails to present a distinct ophthalmoscopic picture, and may be described by saying that the details of the eye-ground are not sharply seen with the aid of any correcting glass.

Causes.—Hyperesthesia and irritation of the retina are usually caused by errors of refraction and anomalies of muscle balance, especially in neurasthenic and hysteric subjects. They also owe their origin to chronic headache, neuralgia, sexual abuses, prolonged fevers, pulmonary disorders, and exposure to bright light. In a series of cases which the author has reported, oxaluria appeared to be the source of trouble.

In some instances of retinal irritation the cause seems to be dependent upon changes in the nasopharynx; for example, engorgement of the septum, associated with myxomatous and hypersensitive spots, vasoparetic and infiltrated turbinals, and secondary changes in the pharynx and larynx. Just as areas of hyperesthesia in these regions may be part of a general neurosis, so, also, they may be both directly and indirectly connected with a hyperesthetic condition of the retina, and the eyes will not grow comfortable until the nasal disease is cured. It is probable that retinal irritation may sometimes be the forerunner of organic change in the optic nerve (Loring).

Treatment.—Spectacles are not a panacea, and although errors of refraction should always be neutralized, the correcting lenses alone do not suffice to relieve the symptoms. General tonics, rest, massage, and all measures calculated to overcome debility or existing neurosis are required. The nasopharynx should be explored. A thorough examination of all organs should be instituted and treatment directed according to the findings. Retinal irritation is apt to be exceedingly stubborn.

Anesthesia of the retina (*neurasthenic* or *nervous asthenopia*), like several other disorders of the retina just considered, should be regarded not as an affection peculiar to the eye, but as one of the symptoms of a complicated neurosis. Very often the condition described in the preceding paragraph and the present affection are closely allied, and with neurasthenic asthenopia there may be marked hyperesthesia and irritation of the retina. On the other hand, such appearances may be entirely absent.

The *subjective symptoms* of this condition have been arranged by Wilbrand and others as follows: Headache, particularly throbbing in the brow and temples, occipital distress, pain in the back of the neck and spine, vertigo, muscæ volitantes, defective accommodation, intolerance of light, and improvement in vision in the dusk and through tinted glasses. Any attempt at concentrated vision is followed by a rapid disappearance from view of the object which is to be fixed. There are diminution of central vision, sudden attacks of obscuration of vision, processions of scotomas, visual hallucinations, lack of fixation of the optical memory images, persistent and confusing after-images, colored vision—for example, erythropsia—and a red appearance of the pages of a book, the letters of which seem to be green.

In this affection peculiar alterations of the visual field, the so-called fatigue contractions, appear. The following forms have been described: The *shifting* or *displacement type*, originally investigated by Förster,

in which the visual fields differ according as the examination is conducted from the temporal side to the nasal, or from the nasal side to the temporal, the contraction being pronounced on the nasal side in the former, and on the temporal side in the latter; the *exhaustion type* of Wilbrand, in which the test-object is moved from the temporal side to the nasal, and from the nasal side to the temporal, several times in succession, across the entire width of the perimeter—indeed, as often as the field continues to diminish; *unstable concentric limitation*—that is, a field which is constantly changing during examination; the *exhaustion-spiral type*, in which the tracing of the visual field appears coiled like a watch-spring, in consequence of its limits becoming concentrically smaller; and, finally, the *recuperation-extension type*, in which the restricted field may extend during rest or by a strong effort of will.

The "oscillating field" described by Wilbrand and O. Koenig may also be found, in which the object disappears and reappears several times in the same meridian, and in which a similar oscillation occurs with colored test-objects. Such fields are not only encountered in so-called nervous asthenopia, but with the retinal exhaustion which is found in a variety of conditions.

Patients thus affected are, for the most part, women, often the subjects of ovarian and uterine diseases, neurasthenia, hysteria, and chlorosis. It is not an uncommon affection in children between the ages of nine and fifteen, in whom, in addition to reduction of central visual acuteness, there is marked contraction of the visual field. Pure types of retinal asthenopia are also seen in men.

Treatment.—This should include all suitable general measures, and not infrequently a "rest-cure," namely, rest with seclusion, forced feeding, massage, and electricity.

Although tinted glasses are recommended, they are not always advisable, lest the affected eyes become too much accustomed to the dull light afforded through such protection. Any error of refraction should be corrected, but spectacles and treatment designed to relieve imbalance of the ocular muscles are usually not alone sufficient to cure these patients. It should be remembered, however, that errors of refraction are often the source of the trouble, and that they must always be thoroughly and carefully corrected if good results are to be obtained. The neglect of this part of the treatment has been the origin of many cases of chronic invalidism.

Cyanosis of the Retina.—This name is applied to an ophthalmoscopic picture seen in patients with congenital heart disease and general cyanosis. The vessels of the fundus are dilated, especially the veins, which may be greatly distended and tortuous. They carry blood much darker than is normal, and the arteries resemble in color the ordinary retinal veins. Small hemorrhages near the disk and larger ones in the macula may be present; sometimes vision is normal; sometimes it is greatly reduced. The ophthalmoscopic appearances of the affection were first described by H. Knapp; they have been studied and depicted in this country by Posey, H. H. Tyson, and T. B. Holloway. In

cyanotic polycythemia the veins of the fundus are greatly enlarged and are very dark colored; the arteries are not materially changed. Retinal hemorrhages may be present (E. Jackson, Parker).

Retinitis.—Under the general term *retinitis* are included the various types of inflammation of the retina.

Varieties and Causes.—Retinitis, like iritis and choroiditis, may depend upon constitutional disorders, altered states of the blood and blood-vessels, infections, auto-intoxication, toxins and traumatisms, or be due to an extension of a diseased process from an inflamed iris, ciliary body, or choroid—that is, the retinitis is either *primary* or *secondary*. Retinitis is often classified according to the probable etiology—for example, *syphilitic*, *renal*, *diabetic*, *hemorrhagic*, etc., retinitis. It is further divided, according to its character, into *circumscribed* and *diffuse*, and was formerly separated, according to its supposed pathologic nature, into *serous* and *parenchymatous* retinitis.

Pathology.—In the acute stage of retinitis the retina exhibits edema and infiltration with leukocytes and red blood-corpuscles. White areas are visible, due to fatty degeneration of both nervous and supporting tissues, varicosity and swelling of the nerve-fiber, and to masses of fibrinous exudation in the granular and nuclear layers. The blood-vessels are thickened, often obliterated, and the supporting tissue hypertropied. In the later stages of atrophy the retina consists of a connective-tissue network which contains many pigment cells; the nervous elements disappear, and the blood-vessels are converted into solid cords. In brief, as Ginsberg summarizes the matter, the changes which present themselves for consideration include edema and exudation, hemorrhage, and small-celled infiltration; proliferation of the neuroglia and the vessel wall connective tissue; degeneration of the retinal elements, the vessels, and the neurogliar tissue; and pigmentation.

Symptoms.—Certain *objective* and *subjective* symptoms are present in most of the forms of retinitis.

1. *Loss in the Transparency of the Retina.*—This may manifest itself as a faint, diffuse haze, a circumscribed opacity and swelling, or as streaks of white infiltration, especially along the lines of the larger vessels.

2. *Areas of Exudation.*—These are an advanced stage of the condition just described. They appear as white spots, sometimes discrete, sometimes confluent, or as patches of bluish-gray, buff, or yellowish color. They should be differentiated from the shining white plaques due to atrophy of the choroid by their softer tone, their situation, and because there is an absence of accumulation of choroidal pigment. They may be present anywhere in the retina or localized in the macular region.

3. *Tortuosity of the Vessels and Change in Their Caliber.*—The veins are darker than normal, unduly wavy in outline, or positively lengthened in their course. The arteries may not be materially changed, but the finer transverse branches are often very tortuous, and both sets of vessels are liable to displacement from their normal level as they cross

areas of thickening, or to partial obscuration by the puffy and infiltrated retina. Many vessels invisible in health become injected in retinitis and form a fine red striation, passing from the nerve-head. Pulsation of the vessels is readily induced by pressure.

4. *Hemorrhages.*—These occur either in the fiber-layer or the deeper portions of the retina. The presence of retinal hemorrhage alone, however, does not indicate the existence of inflammation, as it may occur quite independently of retinitis.

If the hemorrhage is in the nerve-fiber layer, it usually assumes a *flame-shape*, with frayed or feathery edges; if its situation is in the deeper layers, it has a cleaner-cut border and more rounded shape.

5. *Changes in the Nerve-head.*—More or less change in the optic papilla is present: undue redness, loss of the distinctness of its margins, obscuration by the swollen and puffy retinal fibers, or, finally, positive inflammation or neuritis. Atrophy of the disk is commonly present after severe retinitis.

6. *Pigmentation.*—Black spots of pigment mark the situation of former retinal hemorrhages. Pigment in the retina, like hemorrhages, although in many instances a sequence of retinitis, is of itself not necessarily a symptom of inflammation of this membrane.

The difference between pigment in the retina and in the choroid has been described on page 345.

7. *Atrophy of the Retina.*—This, like atrophy of the choroid, may indicate a former hemorrhage or an area of inflammation. All the retinal layers, as well as the choroid, may be involved, exposing a white patch of sclera (*atrophic choroidoretinitis*), or only the superficial layers may be affected, and the spot may be marked by a permanent whitish or yellowish opacity. Contraction of the vessels and white tissue along their coats are often seen after retinitis.

In addition to the ophthalmoscopic signs there are:

1. *Change in Visual Acuteness.*—Central vision is *diminished* in direct proportion to the severity of the case and the situation of the inflammatory action.

2. *Change in the Field of Vision.*—This may be irregularly or concentrically contracted, or scotomas may appear in its center.

3. *Distortion of Vision.*—This occurs under several forms: (*a*) Objects appear to be reduced in size if the retinal elements are spread apart (*micropsia*); (*b*) objects appear to be increased in size if the retinal elements are crowded together (*macropsia*); (*c*) objects appear to undergo change in their contour or shape (*metamorphopsia*). Vertically placed parallel lines, on the one hand, appear to be bulged outward, and, on the other, to be bent inward. Fine parallel lines may appear *wavy* to a normal eye. Retinal metamorphopsia is often associated with a scotoma.

4. *Pain and Photophobia.*—Acute pain is almost always absent, even in violent forms of retinal inflammation; indeed, it is much more likely to be present in the less pronounced grades.

Usually the sensation is one of discomfort rather than of actual

pain. Photophobia may or may not be present. It is never a marked sign, although comfort ensues from the use of tinted glasses.

The **diagnosis** of retinitis depends upon the essential symptom of the disease—opacity or loss of transparency in the retina. All the other symptoms which may be present—exudation, hemorrhages, pigmentation, and atrophy—help to make up the clinical characteristics of the various types, but in themselves are not diagnostic of inflammation of this membrane.

Much diagnostic aid is obtained by noting the effect of the disease upon vision, especially under the influence of diminished illumination, and when acuteness of sight fails quite out of proportion to the amount of the light reduction, the student should at once be upon his guard. Investigation of the light-sense in the manner already described (see page 66) is important. If the coarse changes detailed in the general symptom-grouping are present, the picture is readily interpreted.

Course, Complications, and Prognosis.—The course of a retinitis, like any other inflammation, may be *acute* or *chronic*, and its progress of long or short duration. When the retina and choroid are simultaneously inflamed, a common complication is change in the vitreous (*vitreous opacities*), and an almost constant association is inflammation of the optic papilla, leading to atrophy in prolonged cases (*retinitic atrophy*).

The *prognosis* may be favorable, grave, or positively fatal, depending upon the extent of the inflammation, its situation in the inner or outer layers of the retina, and the cause. Before giving a prognosis the surgeon must always attempt to estimate the extent of the permanent disability which is likely to remain in the form of atrophy of the membrane or secondary changes in the papilla. Other things being equal, the prognosis of syphilitic retinitis is the most favorable.

Treatment.—This, in general terms, demands perfect rest for the inflamed organ, and therefore atropin mydriasis is often desirable. In sthenic cases, in the early stages, blood-letting from the temple has been recommended.

The remedies most likely to afford relief are the various forms of mercury, iodid and bromid of potassium, pilocarpin, and electric-cabinet diaphoresis and Turkish baths. Special methods of treatment are reserved for the sections devoted to the several clinical varieties.

Types of Retinitis.—As an introduction to the special varieties of retinitis which will presently be considered, it serves a useful clinical purpose to refer to two types of retinitis formerly described under the names *serous* and *parenchymatous retinitis*. The first type, also called *retinitis simplex*, *diffuse retinitis*, and *edema of the retina*, is a condition characterized by an infiltration, especially of the nerve-fiber and ganglionic layer of the retina, causing opacity, together with hyperemia, most marked in the veins.

The opacity varies from a delicate veiling to a decided gray-white opacity, most noticeable around the nerve-head, the margins of which are veiled or hidden. From this point the grayish opacity shades out

into the surrounding retina. The disk is not necessarily swollen: it may be simply hidden by the infiltrated tissue, or, if this is not marked, it is very red and its edges obscured by the radiation of finely injected capillaries from its margins. The veins are dark, fuller than normal, tortuous, and often partly covered by the swollen tissue; the arteries are not much changed in size, unless perchance they may be reduced in caliber by compression. Hemorrhages are rare, and exudations in the macular region are uncommon.

There are no external signs of this form of inflammation. Both direct and indirect vision are affected, the former being "foggy," the latter concentrically contracted.

The second type, also called *deep retinitis*, includes those forms of retinitis in which, in addition to edematous infiltration, opacity of the retina, and venous hyperemia, there are pronounced cellular infiltration and structural change, leading finally to atrophy of the elements.

Exudations of yellowish or gray color are visible, occurring in patches throughout the eye-ground, and often localized in a characteristic manner in the macula. Small hemorrhages are commonly present, and the morbid processes may attack the sheaths of the vessels, causing thickening and hypertrophy.

There are no diagnostic external manifestations. Deeply seated pain of a dull, aching character may be present. Vision is often much disturbed, varying from a mere fogginess of the outlines of objects to an almost absolute loss of sight. Contraction of the field of vision and positive scotomas are demonstrable, and the phenomena of distortion of objects are apparent. The disease may be circumscribed or diffuse, and localized in the external or internal layers, or affect both of these and also involve the choroid.

The **prognosis** of the second variety is always grave, and although in certain cases absorption of the products is possible, compression and atrophy of the nervous elements must result in most instances. Independently of the fact that so-called serous retinitis may be the initial change of other forms presently to be described, it has been ascribed to cold, to undue light and heat, to toxins, infections, and to the influence of refractive error in eyes worked under the disadvantage of imperfect illumination. The other type depends, as a rule, upon various constitutional disorders, or occurs in association with other diseases of the eye.

Partaking of the nature of one or the other of these forms there are certain clinical types:

Syphilitic Retinitis.—The syphilitic forms of retinal inflammation have been divided by Alexander into: (1) *Choroidoretinitis;* (2) *simple syphilitic retinitis;* (3) *retinitis with exudations;* (4) *retinitis with hemorrhages;* and (5) *central relapsing retinitis.*

The *first form* in the opinion of many authors is really a disease of the choroid, and the pathologic changes of cellular infiltration, exudation, atrophy, and proliferation of the pigment epithelium are found in the choroid, between the choroid and retina, and in the adjacent retinal layers. G. Nagel, however, points out that in specific *retinochoroiditis*

there are changes in the retinal vessels—that is, a *syphilitic endarteritis*. The pigment changes are produced by wandering and proliferation of the retinal pigment. The choroid is markedly altered; sometimes the choriocapillaris completely disappears. In other words, the retinitis does not depend exclusively upon a choroiditis, nor does the contrary relationship hold good.

The following signs are visible: Opacity of the vitreous, especially in the posterior portion, which resolves itself into fine points or dust-like particles, and stretches out to the periphery like a cloud; loss of transparency of the retina surrounding the nerve-head, which may be unduly hyperemic, and on account of the fine opacity in the vitreous may give the impression that it is swollen; numerous yellowish or white spots of

Fig. 192.—Syphilitic retinitis.

exudation bounded by pigment beneath the vessels of the retina in the periphery of the eye-grounds, and white spots in the macula; and, finally, occasional participation of the iris and posterior layer of the cornea.

The *subjective* symptoms are: Depreciation of central vision, very marked in the later stages; night-blindness and great lessening of visual acuteness under weak illumination; irregular and concentric contraction of the visual field and the formation of ring scotomas as well as positive scotomas in the center of the field; and shimmerings, dancing spots and circles (photopsias), and distortion of objects in the form of micropsia and metamorphopsia due to separation of the rods and cones by the effusion.

In the *second form* there appears to be a localization of the disease in the retina, particularly its inner layers, and this tissue is, as Schöbl expresses it, first, and often alone, selected by the syphilitic poison. The ophthalmoscope reveals a gray opacity surrounding the nerve entrance and stretching out in lines along the vessels; the papilla is discolored, cloudy, and has been compared to a yellowish-red, oval body seen through a covering of fog. The veins are darker than normal; the arteries usually are not materially changed.

Other objective symptoms in syphilitic retinal disease are floating vitreous opacities, exudations along the lines of the vessels (*retinitis with exudations, perivasculitis*), and extravasations of blood, usually round in shape, attributed to disease of the vessel walls (*endarteritis*) or to the formation of thrombi (*retinitis with hemorrhage*). Hemorrhages in syphilitic retinitis, however, are of comparatively rare occurrence.

According to Haab, *syphilitic endarteritis* is a comparatively rare disease, and may present the following lesions: Visible opacity of the walls of the arteries and rarely of the veins, so that the blood-columns of the diseased vessels are accompanied with lateral white lines; almost invisible disease of the vessel walls, manifesting itself, as in senile sclerosis, by a narrowing of the blood-columns, and sometimes associated with extravasations of blood; an opacity corresponding to that caused by an obstruction of the central artery or one of its branches, and appearing as a gray-white or a milky area, with ill-defined edges, in which at times considerable hemorrhage may take place; and groups of circumscribed white patches somewhat resembling those seen in albuminuria.

Date of Occurrence.—Diffuse syphilitic retinitis may occur in congenital and acquired syphilis. In the acquired form of the disease it appears from one to two years after infection, sometimes as early as the sixth month, and is found in about 8 per cent. of the cases (Alexander). One eye alone may be affected, but usually after several months the second eye is also involved.

True retinitis must not be confounded with the so-called "retinal irritation" commonly seen in association with iritis, and the symptoms of which have been described under Hyperemia. Retinitis, however, may accompany or follow iritis.

Course and Prognosis.—Although the onset of syphilitic retinitis may be sudden, the course is essentially chronic.

The *prognosis* largely depends upon the stage at which treatment is begun and the vigor of the measures employed. Delayed or neglected treatment may lead to the grave consequences of extensive choroiditis, pigmentary degeneration in the retina, and atrophy of the optic disk. Even in favorable circumstances improvement may be temporary and many stubborn relapses occur.

Treatment.—The same constitutional measures recommended in the treatment of syphilitic iritis (see page 309) are indicated, and, in so far as mercurials are concerned, should be vigorously employed. Concerning the value of salvarsan in syphilitic retinitis, it may be said that successes have been reported even in the presence of decided endar-

teritis, and in the author's experience the use of neosalvarsan in the manner already described has been followed by admirable results. Usually a mydriatic is advisable, and in any event dark glasses may be worn.

Central relapsing retinitis (*retinitis macularis*) belongs to the late manifestations of syphilis, and appears in the form of a gray or yellow area in the macula, or as numerous small yellow or yellowish-white spots and pigment dots, or as a diffuse opacity of this region. The papilla and its surroundings are unaffected. It is a rare form of syphilitic retinitis, stubborn in its character, and prone to relapse.

Hereditary Syphilitic Choroidoretinitis.—Various types of hereditary syphilitic affections of the retina and choroid occur, and they

Fig. 193.—Appearances of the eye-ground in hereditary syphilis (from a patient in the University Hospital).

have been particularly well described and depicted by Haab and Sidler-Huguenin. Whether the primary seat of the disease in these cases is in the retina or in the choroid has not, in Haab's opinion, been definitely settled. According to these authors, some of the following types may be encountered: (1) The periphery of the eye-ground presents a somewhat leaden color and contains black circular and triangular patches of pigment. The remainder of the fundus is thickly covered with reddish-yellow spots placed upon a dotted brownish-black surface. Occasionally these lesions are not extensive and cover only certain portions of the fundus, especially the periphery. (2) Chiefly in the periphery of the eye-ground roundish black foci of pigment, discrete and confluent, are evident, interspersed with linear and circular yellowish patches. The

lesions are not infrequently seen after the subsidence of interstitial keratitis. (3) In place of gray and black lesions, whitish circular or confluent patches may be found in the periphery of the fundus. Sometimes these types are mixed, and in some severe cases there are coarse choroidoretinitis, diseased retinal vessels, and atrophy of the optic nerve. There may be diminution of central vision, contraction of the field of vision, and night-blindness, symptoms which are absent in mild manifestations of the disease.

Metastatic Retinitis (*Septic Retinitis of Roth*).—This term has been applied to an affection especially seen in surgical pyemia and puerperal septicemia, and is characterized by small, circumscribed white spots near the papilla and in the macular region. Usually both eyes are involved, and numerous small hemorrhages may be seen. These spots are due to fatty degeneration of the capillaries and infiltration of the retinal fibers, caused by the infectious emboli in the vessels. Microorganisms have been demonstrated in the lesions and in the retinal vessels.

The spread of the inflammation to the uveal tract and the relation of this condition to purulent *metastatic ophthalmitis* (choroiditis) has been described on page 358. This condition is also sometimes called *embolic panophthalmitis* or *endogenous ophthalmitis.*

An independent or *primary suppurative retinitis* may be caused by injury, that is, by a penetrating foreign body (see also page 294).

Treatment.—The prognosis and treatment of suppurative retinitis does not materially differ from that recorded in connection with metastatic ophthalmitis. Occasional recoveries are recorded with preservation of eyesight.

Hemorrhagic Retinitis.—The presence of hemorrhages in the retina does not imply the coexistence of retinitis; only if signs of inflammation are added is the term "hemorrhagic retinitis" justified.

In a typical case the appearances are as follows: Swelling of the papilla, its edges being clouded or hidden by an opaque infiltration of the surrounding retina; darkly tortuous and distended veins, but small arteries; and numerous hemorrhages, linear, flame shaped, irregular, or round in shape.

The size, number, diffusion, and localization of the hemorrhages vary. Thus, they may be everywhere throughout the eye-ground, or grouped, especially in the macular region or around the papilla. If white spots are present as the result of degeneration after absorption of the blood, the appearances may closely resemble those seen in so-called albuminuric retinitis, which, indeed, may be one of the types of hemorrhagic retinitis.

Causes.—Hemorrhagic retinitis occurs with diseases of the heart and of the blood-vessels—*e. g.*, hypertrophy, aneurysm, and endarteritis; in suppressed menstruation; at the climacteric; and in a variety of general and local diseases, sometimes presenting types presently to be described under special clinical designations. More rarely, retinitis with hemorrhages is caused by secondary syphilis.

The hemorrhages may be due to rupture of retinal vessels whose coats have become degenerated—in other words, they depend upon endarteritis; but recent investigations show that in many cases, although the arteries may be diseased, there is even more extensive change in venous coats, and there may be thrombosis of the central vein. The disease is often monocular. The connection between degeneration of the blood-vessels and chronic inflammation of the inner layers of the retina is an intimate one. To all inflammation of the layers of the retina the term *angiopathic retinitis* is applied by Wildbrand and Saenger. (For further consideration of this subject, see Hemorrhage in the Retina, p. 451.)

Prognosis.—This is unfavorable because the ocular condition may indicate a grave vascular or cardiac malady, and may be the forerunner of extravasations in vital centers. Sight may be seriously impaired. Secondary changes in the retina and optic nerve are likely to follow; glaucoma frequently results.

Treatment.—The therapeutic measures must be governed by the general condition. F. R. Cross recommends subconjunctival blood-letting, and wet-cupping the temple has been advised. Often mercury, iodid of potassium, and iodid of sodium are indicated, with or without cardiac sedatives, and diaphoresis may be required.

Albuminuric Retinitis (*Renal Retinitis; Papilloretinitis; Retinitis of Bright's Disease*).—**Symptoms.**—In a typical case, beginning in the macula or its immediate neighborhood, and continuing to be most numerous in this region, variously shaped and placed white spots appear. These at first may be small, discrete, and sharply separated, but later, or under other conditions, they form a somewhat *star-shaped figure*, the rays of which surround the fovea, but for the most part do not involve it. Occasionally, instead of a stellate arrangement, the white spots and lines, somewhat radially placed like spokes in a wheel, affect this neighborhood in part, but do not completely encircle it.

At some distance from the papilla, and often surrounding it, larger yellowish-white or white spots are seen, which may coalesce and form a ring-shaped zone around the nerve-head broader than its own diameter. This striking, wide white area has been compared to snow, and designated "the snowbank appearance of the retina."

Other features, but, unlike the white spots, having no pathognomonic appearances, are the *hemorrhages*. They may be linear, flame shaped, or round, or mere flecks scattered here and there, and found with difficulty, or they constitute large, dark-red extravasations. Moreover, they are not constant like the white spots, but at times disappear, leaving white marks which denote their former situation. Sometimes they occur in great numbers, like fresh explosions. To a certain extent they are indications of the violence of the disease.

The blood-vessels may run over the white plaques, or may be buried in the swollen retina. Sometimes a vessel disappears beneath the infiltration, to reappear at some distance beyond. The veins are dark and often tortuous; the arteries, as in other forms of retinitis, are not mate-

rially altered in size. In the later stages the vessels exhibit lack of transparency of their walls, in the form of white tissue along the sheaths, or they are actually converted into white strings.

Finally, the optic papilla and its immediate surroundings may be intensely hyperemic, or a swelling of the nerve-head occurs, quite indistinguishable from that of *optic neuritis*, or *choked disk*, as it is seen in tumor of the brain. In any circumstances the edge of the papilla is clouded, but not necessarily swollen, the surrounding retina finely clouded, and traversed with numerous radiating injected lines, like those described in other types of retinitis. Quite commonly the changes in the papilla directly join the band of fatty infiltration already described, surrounding the end of the optic nerve.

The chief, in fact the only, *subjective* symptom is depreciation of vision, which may vary from a slight and gradual impairment to complete blindness. It is a well-known fact that Bright's disease is often discovered by an ophthalmoscopic examination, the patient being ignorant of the fact that he is the subject of serious organic malady. The visual field may be altered according to the situation of the retinal lesions, and may contain blue-blind areas. According to Gerhardt, blue-blindness may be a sign of contracted kidney, and Simon maintains that violet-blindness is not uncommon in connection with albuminuric retinitis.

Forms of the Disease.—Two varieties have been recognized—an *inflammatory* or *exudative* and a *degenerative* type. Often the two are combined.

The former may be present as violent *neuroretinitis* from the beginning, or it may start as a degenerative type and assume inflammatory action. The latter begins without inflammatory changes, the white spots are small, often quite minute, and separated by comparatively normal areas, and the hemorrhages, if present, are inconspicuous, being confined largely to the nerve-fiber layer. The arteries are sclerotic, the veins dark, and the disk, in the early stages, blurred and indistinct, but there is no peripapillary zone of white exudation and no macular figure. If hemorrhages are the most conspicuous feature of the disease, the term *hemorrhagic* is applied; if the changes are almost wholly confined to the optic papilla, the *neuritic* or *papillitic* type is developed. Samuel West draws a sharp distinction between degenerative and exudative albuminuric retinitis, and associates the former with granular kidney and the latter with parenchymatous nephritis. The exudative variety is inflammatory and probably toxic in origin; the degenerative depends on vascular changes.

Often small hemorrhages and comparatively insignificant dots in the macula may be the signs of renal retinitis, and consequently of renal disease. Indeed, the so-called typical renal retinitis is not so frequently encountered as the less elaborately produced lesions of this affection. Among early signs of renal retinitis are changes in the capillary circulation, and dilatation and tortuosity of the small vessels around the macula, while the nerve-head assumes a congested, brick-red color. In every

PLATE IV.

Albuminuric retinitis; star-shaped figure in the macula; the circulation in the distended veins impeded where the latter are crossed by the arteries which are undergoing sclerotic changes.

case of retinal disease the urine should be frequently and thoroughly examined.

Causes, Date of Occurrence, and Frequency.—While in general terms Bright's disease is the cause of the retinitis which bears its name, it most frequently occurs with chronic interstitial nephritis. It may also be caused by chronic parenchymatous nephritis, especially in the so-called inflammatory form. Naturally, the secondary contracted kidney, which is a sequence of large white kidney, may be associated with retinitis, and this is also true of amyloid disease of the kidney. The retinitis occurring with pregnancy is usually ascribed to albuminuria, but is probably due to the same substances which cause the albuminuria, the eclampsia, vomiting, etc., namely, toxemic products in the circulation. Retinitis may arise in the course of a scarlatinal nephritis.

Usually both eyes are involved, but *unilateral albuminuric retinitis* is not a rarity (Knies), a certain percentage of cases maintaining monocular retinal lesions until death. In another large percentage of cases the unilateral character of the affection is temporary, both eyes ultimately becoming affected. In general terms it is probable that the renal disease must be present for some months before retinal lesions appear. The age at which patients are attacked is usually stated to vary from thirty to sixty, the most prolific single decade, according to Nettleship, being from fifty to sixty. It is comparatively rare before the twenty-fifth year, but children and young persons are not exempt.

About twice as many cases of renal retinitis occur in men as in women. If there is decided hyaline thickening of the retinal arteries, an early stage of granular kidney may be suspected, especially if the patient is comparatively young (Nettleship). The recorded percentage of retinitis in renal disease varies from 9 to 33. In the author's experience fully 25 per cent. of patients with chronic Bright's disease, as he has examined them in general hospitals, have been affected by various forms of retinitis, but if these statistics should include not only the cases of so-called typical retinitis, but also those of comparatively insignificant lesions, consisting chiefly of alterations in the walls of the retinal vessels and blurring of the disk, this percentage would be considerably higher.

Course, Pathology, and Prognosis.—The course of typical renal retinitis has been divided into the stage of hyperemia of the papilla, opacity of the retina, and hemorrhages; the stage of fatty degeneration; and the stage of retrograde metamorphosis and atrophy.

The white spots may subside, but rarely disappear entirely, the macular changes being most permanent. Discoloration and atrophy of the papilla, contraction of the vessels and the formation of white tissue along their walls, and pigment changes in the retina finally result.

The *pathologic changes* are found chiefly in the macular region and in a zone surrounding the nerve. The retina is thickened by the presence of the so-called inflammatory edema and by hypertrophy of its nervous and supporting tissue. The glistening spots in the macular region are due to a fatty degeneration of the exudation and of the retinal

elements. Their star-shaped arrangement depends upon the oblique direction of the fibers of Müller in this position. Many fatty granular cells and deposits of coagulated fibrin are seen, particularly in the nuclear layers. Hemorrhages are present, but not necessarily a pronounced feature. In the early stages the vessels show thickening of the adventitia, and later pronounced hyaline change and proliferation of the lining endothelium. The nerve in many cases is swollen by the inflammatory edema. The same causes which originate disease of the blood-vessels of the kidney originate also the alterations in the retinal vessels, and to these alterations the chief rôle must be ascribed in causing the various types of retinal lesions. Indeed, some authorities maintain that so-called albuminuric retinitis is entirely the outcome of disturbances in the circulation, that is, depends on arterio- and phlebosclerosis and their sequels. Sclerotic changes in the choroid vessels are also present.

Detachment of the retina, hemorrhage into the vitreous, embolism and thrombosis of the vessels, extravasations into the choroid, and glaucoma may be *complications* of this affection. Detachment of the retina is not infrequent, and glaucoma may arise exactly as it does with retinal hemorrhages and retinal angiosclerosis.

The *prognosis*, so far as vision is concerned, depends upon the extent of the lesions and of the involvement of the macula. In general terms it is unfavorable, although fair vision is often retained. Sometimes the exudations practically disappear. In so far as the life of the patient is concerned, albuminuric retinitis is a most unfavorable symptom, and many patients die within two years after its detection, and a considerable percentage within the first year of its development. There are, however, frequent exceptions to the rule, and the records show that patients have lived five, seven, and even a greater number of years after the retinal lesions have appeared, especially if they have been detected early and suitable treatment has been instituted.

Albuminuric Retinitis in Pregnancy.—While the occurrence of albuminuria during pregnancy is not uncommon, varying, according to statistical reports, from 2 to 20 per cent., involvement of the optic nerve and retina, in the form of a neuroretinitis, to which the term *albuminuric retinitis of pregnancy* is usually applied, is much less frequent. The retinitis in this condition may gradually develop, occurs most frequently in primiparæ, and generally in the second half of pregnancy; exceptionally at an earlier period. The ophthalmoscopic signs of this retinitis may not differ from those which are caused by other forms of Bright's disease, and, in general terms, there is a wide-spread neuroretinitis with exudations and hemorrhages. The retinitis of pregnancy has been ascribed to a nephritis which is brought about by this condition, especially a fatty degeneration of the kidney epithelium, and also to toxemic products in the circulation (J. H. Fisher; see also page 435). It may also be caused by an acute nephritis which has developed during the pregnant period, and by an exacerbation of a pre-existing chronic nephritis during the same period.

In the *albuminuric retinitis of pregnancy* the prognosis, in so far as it concerns the vision and the life of the patient, depends upon the duration of gestation. With the termination of pregnancy the inflammatory deposits (the type most often is inflammatory) may subside and good vision may be restored, provided the process has not continued so long that the secondary changes already described have taken place. For this reason the induction of premature labor has been recommended as a therapeutic measure, and if the visual disturbances appear during the first six months of gestation usually the pregnancy should be terminated if sight is to be saved.

Diagnosis.—In wide-spread albuminuric retinitis the changes detailed in the symptom-grouping are striking and in a sense character-

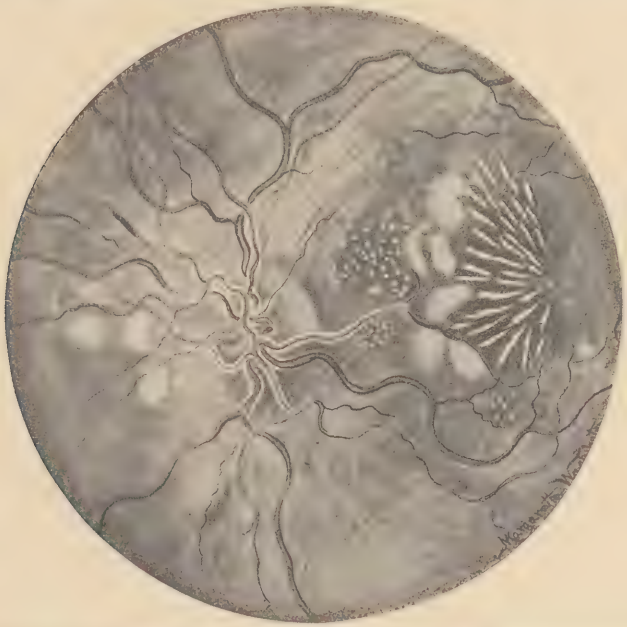


Fig. 194.—Albuminuric retinitis of pregnancy. Colored patient in the University Hospital.

istic, but the so-called typical cases are not as frequent as those in which the lesions are not so evident, and the significance of the retinal disease must be decided by general examination. Even so-called "typical" appearances have been observed in infections—for example, erysipelas and syphilis—independently of nephritis. Thus, Schieck has investigated a certain number of eyes with *macular changes* which were regarded as characteristic of nephritis, and yet the evidence of kidney disease was lacking; similar observations have been made by Wildbrand and Saenger, by Parsons, and by the author.

Neuroretinitis from intracranial disease may simulate this affection, and often only by a careful study of the urine and the general symptoms the diagnosis can be established (see also page 479). The question be-

comes still more complicated if albuminuria is associated with brain tumor.

In glycosuria and leukemia somewhat analogous appearances are found, and again, an examination of the urine, as well as that of the blood, may be necessary before reaching a diagnosis.

The white spots are distinguished from plaques of choroidal atrophy by the absence of pigment heaping. The snowbank appearances differ from retained marrow sheath (see page 469) in that the latter stretches away from the margin of the disk, usually ending in a fan-shaped border, and is unaccompanied by the changes in the macula or by retinal edema. Fine lesions of the choroid in the macular region may be mistaken for somewhat similar retinal changes; but they are more scattered, more yellow in color, usually unassociated with distinct loss of vision, and less liable to assume a stellate or radial appearance.

Treatment.—Local measures are practically of no avail. The case must be managed on the general principles suited to the form of kidney disease which is present. A proper remedy in most cases is iron, usually in the form of the tincture, and often advantageously combined with bichlorid of mercury. Decapsulation of the kidney has been tried without encouraging success. Harvey Cushing has recommended cerebral decompression, because he believes that increased intracranial tension is an important factor in the development of albuminuric retinitis. The author's single experience with this operation in these circumstances was disastrous.

Diabetic Retinitis.—This occurs in several forms. It is always bilateral, but both eyes may not be affected at the same time.

Hirschberg describes two varieties of diabetic retinitis—an *exudative* and a *hemorrhagic* form. In some cases of diabetic retinitis, either with or without hemorrhage, there are wide-spread areas of yellowish-white exudation and fatty change, and these lesions may arrange themselves in zone-like areas, above or below the macula, resembling the so-called circinate retinitis, and may be massed in the central region of the retina. They usually are late manifestations of diabetes, and are seen at a time when gangrene, carbuncle, hemiplegia, and other serious complications of this disorder arise. In any case of diabetes of long duration retinitis is seldom absent, although it may sometimes be difficult to find the lesions, because they are situated in the periphery of the eye-ground. This is especially true if the complication of high myopia, or cataractous lens, is present.

More commonly than in the retinitis of albuminuria, opacities and hemorrhages occur in the vitreous humor, and a condition analogous to proliferating retinitis may arise. To a collection of small white spots and hemorrhages irregularly arranged in the macular region and between it and the disk the name *central punctate diabetic retinitis* has been applied. By some authors this appearance is considered typical of diabetes. The vital prognosis is unfavorable, but not nearly so grave as in albuminuric retinitis.

To a striking ophthalmoscopic picture characterized by a very

light salmon color of the blood in the retinal arteries and veins, which are much enlarged, and by a somewhat light color of the general fundus the name *lipemia retinalis* has been given (Heyl). These appearances are ascribed to the presence of fat in abnormal amounts in the blood. No hemorrhages or exudations develop; in this respect it differs from leukemic retinitis. It is apt to occur in young diabetics and implies a grave prognosis.

Treatment.—There is no local treatment of diabetic retinitis. The discovery of such a condition may lead to the finding of sugar in the urine, but more commonly the patient is already conscious of his disease and is under medicinal and dietetic treatment.

Fig. 195.—Diabetic retinitis; extensive white exudations in the macular region.

Leukemic Retinitis.—The retinal changes seen in splenic leukemia, to which variety of the disease they are almost exclusively confined, affect both eyes, usually one more than its fellow.

The most important ophthalmoscopic appearances are slight swelling of the papilla, pallor of its surface, veiling of its edges, and some opacity of the retina, especially along the lines of the vessels. The latter present a striking appearance. The veins are broad, distended, and of a somewhat rose-red color; the arteries, in contrast, narrow and orange yellow, which color substitutes the ordinary fiery red of the choroid, the vessels of which, if they are visible, present a yellowish-red tint.

Very prominent lesions are white spots with red borders, especially near the equator and in the region of the macula lutea. The spots

vary in size and are often somewhat elevated. They are due to a collection of lymph-corpuscles, and the red border to an extravasation of blood-corpuscles.

On the other hand, retinitis associated with leukemia may not present characteristic appearances, but may consist simply of a diffuse opacity of the retina, or appear in the form of hemorrhagic retinitis. When the yellow spots which have been described develop in the macula they resemble the lesions produced by albuminuria. Indeed, albumin in the urine may be present with leukemia. In any doubtful case a careful blood examination will reveal the true nature of the disease.

Proliferating Retinitis.—This affection is characterized by dense masses of bluish-white or white color, which are developed from the

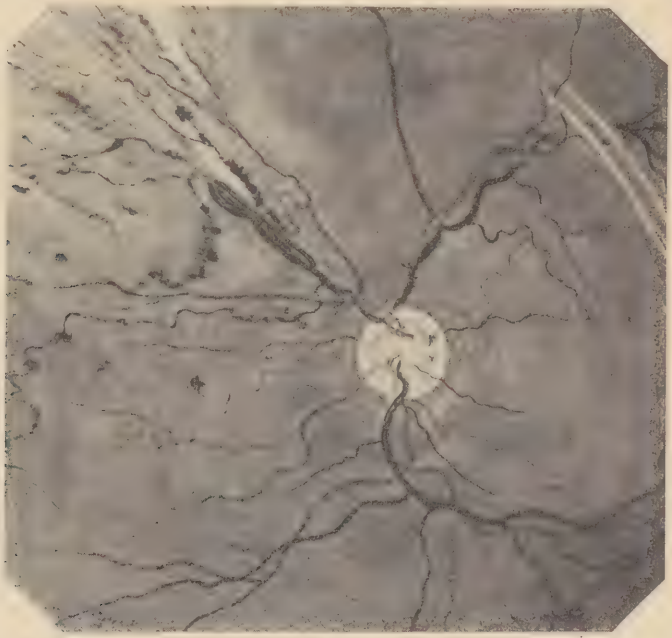


Fig. 196.—Proliferating retinitis in an early stage. Note the beginning formation of membranes extending from retina into vitreous.

retina and stretch out into the vitreous humor. They often cover a considerable portion of the fundus and hide the papilla, which may with difficulty be seen through the intervening spaces. Sometimes the masses follow the course of the blood-vessels, which in part may lie beneath them, and in part pass over them; those which lie above the masses are occasionally newly formed blood-vessels. As complicating circumstances there may be detachment of the retina, opacity of, and hemorrhage into, the vitreous.

According to Weeks, the essential of this disease is the production of membranes which extend from the retina into the vitreous humor, and a fibrinous exudation or hemorrhage must first occur before these

membranes can be formed. This process and that of vascular growths in the vitreous (see page 419), as Marple has well shown, are identical. Two varieties of the affection have been described—syphilitic and traumatic—but, as Axenfeld has shown (see page 418), tuberculosis or its toxin may be the active agent. In these circumstances tuberculin injections should be given a trial. The disease is more common in young than in elderly subjects. Vision is greatly impaired, sometimes totally lost.

Retinitis Circinata.—This name was applied by Fuchs to an affection characterized by a concentric aggregation of slightly raised white spots and lines around the macula. Sometimes the white spots surround the macula after the manner of a wreath; sometimes the arrangement is more like that of an ellipse, one end of which may touch

Fig. 197.—Circinate retinitis (from a patient in the Jefferson Hospital).

the edge of the optic disk, while the other extends beyond the macular region (Lawford). Fuchs regards the white patches as fibrinous exudations which had taken place into the deeper layers of the retina, while de Wecker denied the special character of the disease, which he attributed to fatty degeneration, the result of hemorrhages. Indeed, Amman has shown that the white spots are due to fatty cells clustered where hemorrhages have been. Hemorrhages may accompany the affection, and in one case (Fridenberg) there was a development of new-formed blood-vessels in the retina. The lesions have also been attributed to disease of the smallest macular vessels, especially the arteries (Oeller), and also to the results of a long-standing edema, the size of the circle varying according to the extent of the previous affection (Gunn). Sometimes the disease is essentially chronic and the appearance remains unchanged for years; sometimes it is slowly but surely progressive, and

rarely the ring of exudate may partly or entirely disappear. Such disappearance within one year has been noted by N. Bishop Harman.

Central Punctate Retinitis (*Retinitis Punctata Albescens*).—This type of retinal affection was originally described by Mooren, and, according to him, is characterized by a great number of striæ or spots scattered over the fundus, resembling in color the reflex of the sclera. The retinal vessels are not covered by the spots. The papilla is decidedly gray. While the peripheral field of vision is unaffected, in its center there may be either a relative or a positive scotoma. Vitreous hemorrhages have also been described, and in Hirschberg's cases, who calls the disease *central punctate* and *striated retinitis*, atheromatous changes in the vessels elsewhere in the body were found. Fuchs has called attention to the similarity of this disease to retinitis pigmentosa, inasmuch as it is either congenital or starts in infancy, affects several members of the same family, and may occur in the children of blood relations. Also, there may be night-blindness and contraction of the visual field. As John Griffith has pointed out, it should be regarded as a primary degeneration of the retina and choroid allied to pigmentary degeneration of the retina, and should not be classified as an inflammatory disease. Another type of chronic retinal degeneration is that to which Fuchs gives the name *atrophia gyrata choroideæ et retinæ*, also seen in the children of consanguineous marriages and associated with night-blindness.

Treatment.—This consists in depletion from the temple, and iodid of potassium or other alterative of similar physiologic action.

Retinitis Striata.—Occasionally light or yellowish-white stripes extending from the periphery toward the disk, and sometimes bordered by lines of pigment lying beneath the retinal vessels, are apparent to the ophthalmoscope. To this appearance the name retinitis striata has been given, and while the origin of the affection is not positively known, Holden contends that the stripes are the result of the metamorphosis of retinal hemorrhages, and in this respect are analogous to angioid streaks. On the other hand, it has been contended by L. Caspar that these retinal striations represent the final stages of spontaneously cured detachments of the retina. The author has published the case histories of two patients which appear to confirm this view of the origin of retinitis striata.

Pigmentary Degeneration of the Retina (*Retinitis Pigmentosa*).—Although this affection is usually entitled *retinitis pigmentosa*, the phenomena of inflammation are absent, and it consists of a degeneration of the nerve tissue, associated with great contraction of the blood-vessels and the accumulation and deposition of pigment of well-nigh characteristic form in the substance of the retina.

Symptoms.—The ophthalmoscopic appearances of a typical case are as follows:

(*a*) *Pigmentation.*—The pigment masses frequently assume an appearance resembling bone-corpuscles, and by the union of their processes suggest the Haversian canals. The resemblance of the pigment to bone-

corpuscles is not always evident; the pigment deposits may be round and irregular and simulate the pigment spots of choroiditis, but unlike them they are situated in front of the blood-vessels and are in the inner layers of the retina. By preference, the pigmentary deposits are more marked on the temporal side. They begin in the periphery of the eye-ground, although not usually in the extreme periphery, often lying along the course of the main vessels, which may be in places encrusted by them, and gradually approach the papilla, the macular region remaining for a long time unaffected. A zone midway between the center and far periphery is the favorite seat of pigmentation.

(*b*) "*Wainscoted*" *Fundus.*—A perfect picture of the appearance already described in connection with superficial choroiditis is visible on account of the absorption and decolorization of the retinal pigment epithelium and the exposure of the larger vessels of the choroid. The overlying retina is distinctly gray.

(*c*) *Contraction of the Vessels.*—This is present in both systems. The vessels may be as thin as threads. Often their walls exhibit patches of opacity, and they are accompanied by fine white lines and covered here and there by pigment deposits. Not only are they greatly contracted, but they are apparently diminished in number.

(*d*) *The Changed Nerve-head.*—The color of the papilla, according to the stage of the disease, is of a yellowish-gray, yellowish-red, or waxy tint. It finally becomes dull white and atrophic. Except a slight veiling, its edges are plainly marked.

(*e*) *Opacities of the Media.*—Posterior polar cataract is frequently present, and in the later stages posterior cortical cataract. Opacities in the vitreous are uncommon.

(*f*) *Nystagmus.*—Quite frequently a quick lateral oscillation of the eyeballs, or nystagmus, is present, especially in congenital cases.

The subjective symptoms are:

(*a*) *Depreciation of Central Vision.*—Visual acuteness may be but slightly affected in the earlier stages, although usually the perception of green and red is below the normal (Oliver). Indeed, reasonably good central vision may remain even when the disease is very wide-spread, but it finally sinks with the progress of the affection and, ultimately, blindness results, although this usually does not occur until the expiration of many years.

(*b*) *Contraction of the Field of Vision.*—In the early stages of pigmentary degeneration of the retina the field of vision may be nearly normal in extent, if the illumination is good, although much contracted if the illumination is reduced. Later, the field contracts concentrically, according to the amount of degeneration, and the contraction may be so excessive that only a very small area of the field remains. In rare instances, even with extreme narrowing of the visual field, there is still moderately good central vision, and the patient may read by fixing a single word at a time. Finally, the contraction goes on to complete blindness. As the *extreme* periphery of the retina is often free from pigmentation in the earliest stages of the disease, when the equa-

torial region is already involved, the periphery of the visual field may be intact, but between it and the preserved central field there is a blind zone; that is, an *annular* or *ring scotoma*. According to M. L. Hepburn, the earliest manifestation of primary degeneration is this ring scotoma. At first it is incomplete and represents the loss of function in the intermediary zone of the retina. Later other portions of the retina degenerate in regular order, the fixation point being the last to disappear. This is in accord with the author's experience. On the other hand, W. T. Shoemaker, as the result of his investigation of pigmentary degeneration of the retina in deaf mutes (17 cases), concludes that ring scotoma in this disease is a rare field defect.

(*c*) *Night-blindness.*—Often this is the first symptom which calls attention to the case. The patient is uncertain in his movements and stumbles as soon as twilight begins, becoming quite helpless in the dark. Night-blindness is not always present, and in rare instances

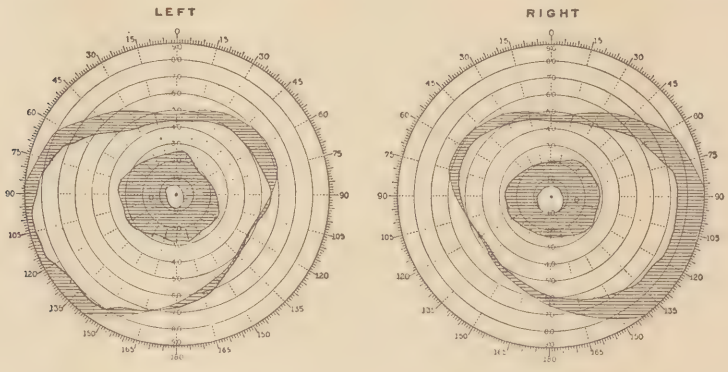


Fig. 198.—Ring scotomas in pigmentary degeneration of the retina.

diminished light is a relief to the patient. Such a condition is due to retinal hyperesthesia.

Atypical Varieties.—The pigment may be massed in the macular region; the central vision is much affected, and a scotoma appears around the point of fixation. In other instances the pigment is scattered all over the fundus in irregular masses, and is associated with clear, shining spots lying beneath the retinal vessels. Cases occur presenting the usual subjective symptoms, but without the accumulation of pigment—really forms of *sclerosis of the retina without the formation of pigment*—and a few instances, associated with a broad peripheral zone of choroidal atrophy, have been described. Finally, in rare instances, retinitis pigmentosa may be complicated with chronic glaucoma, the retinal affection probably antedating the glaucoma (Bellarminoff, Mandelstamm). Holloway has described pigmentary degeneration of the retina in association with albuminuric neuroretinitis.

Causes.—The disease is markedly hereditary. Simeon Snell has reported the history of this affection in five generations, 28 of the 67

descendants being affected. Consanguinity of the parents of the patient has been found in a certain number of the cases; indeed, the disease has been attributed to this cause alone. Nettleship's results are as follows: Heredity without consanguinity in 23.5 per cent., consanguinity without heredity in 23 per cent., and heredity combined with consanguinity in 3 to 4 per cent. Hereditary syphilis has been suggested as a possible cause of retinitis pigmentosa, but this etiology has not been proved. The affection is found among deaf mutes, idiots, and epileptics, and in this sense is connected with morbid states of the nervous system. Very often no cause can be assigned. The disease is either congenital or begins in childhood. According to W. T. Shoe-

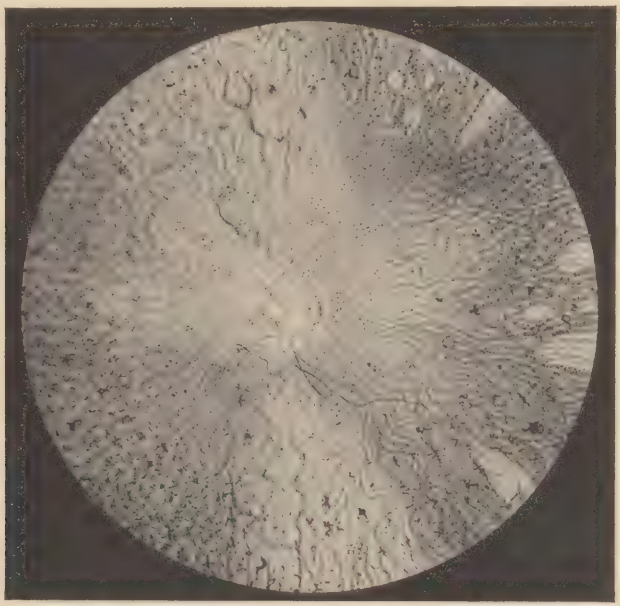


Fig. 199.—Pigmentary degeneration of the retina; marked exposure of vessels of choroid (from a patient in the University Hospital).

maker, it is always congenital in origin, no matter how late its manifestations are evident.

Pathology.—The degenerative process begins in the outer layers of the retina, which becomes adherent to the choroid. The rods and cones disappear, the blood-vessels are sclerosed, and their lumens contracted. Later the sclerotic process reaches the layer of ganglion cells and the nerve-fibers. There is a marked infiltration of pigment cells along the thickened vessels. The optic nerve atrophies and may contain hyaline masses similar to those in the lamina vitrea of the choroid. Wagenmann believes that the primary lesion is a sclerosis of the vessels of the choroid, and W. T. Shoemaker concludes that within the eyeball the primary tissue involved is the choroid. Gonin and Nettleship attribute the primary degeneration to deficient blood-supply owing to

obstruction to the blood-current in the choriocapillaris. The beginning of the disease has also been placed in the pigment epithelium. The affection is always bilateral.

Diagnosis.—Retinitis pigmentosa may be distinguished from disseminated choroiditis by the difference in the pigmentation of the two diseases.

Its differential diagnosis from certain types of pigmented retinochoroiditis seen in acquired syphilis is difficult, especially when the latter manifest themselves in the form of atrophy of the retina and a gathering of pigment spots, beneath which the exposed choroidal vessels are visible. In retinochoroiditis, however, the pigment spots do not have the characteristic form; they are much scattered, and do not follow or cover the blood-vessels; besides, vitreous opacities, which are comparatively rare in pigmentary degeneration of the retina, are usually present. The visual field should lend aid in diagnosis, as in choroidal disease the characteristic feature is the patchy nature of the scotomas (M. L. Hepburn).

A patient with night-blindness, or seen stumbling about during the twilight, should be subjected to a careful examination of the periphery of the eye-ground, if necessary, after dilatation of the pupil, because occasionally the pigment is confined to this region and might be overlooked by a careless observer.

Course and Prognosis.—Pigmentary degeneration of the retina progresses steadily onward with ever-increasing contraction of the field of vision, until finally, usually by middle life, sight has been obliterated, with, perhaps, the exception of a slight eccentric preservation of the field. According to Nettleship, the age at which blindness becomes complete is variable, and, with rare exceptions, occurs only after the thirtieth to the thirty-fifth year of life; more usually after sixty years. The prognosis is, hence, nearly always unfavorable in all circumstances and in spite of all known endeavors to modify the course of the disease. Occasionally, when the pigment accumulation has advanced far over the retina, but the macula is still free, the disease remains stationary for long periods of time, and good vision within the narrow field continues.

Treatment.—This is of little avail. Strychnin in full doses, especially by the hypodermic method, has been recommended. If there is any suspicion of syphilitic taint, the usual remedies are indicated. Galvanism has been tried, and under its influence, it is stated, the progressive contraction of the field of vision has been stayed, although no improvement in the acuteness of central sight was obtained. It certainly should be given a trial in every case.

Sometimes a peculiar *pigmentation of the retina* is encountered which has been mistaken for a type of retinitis pigmentosa, with which disease, however, it has no alliance. The pigment changes occupy a sector of the eyeground, and consist of collections of black or chocolate-brown spots, with a strong tendency to group formation. The spots may be round, oval, and often suggest diminutive bunches of grapes. This appearance was figured by Jaeger, has been described by Juler, Stephen-

son, and others, and, quite recently, elaborately by Hoeg. It is doubtless a congenital condition; the function of the eye is undisturbed.

Detachment of the Retina (*Ablatio Retinæ; Amotio Retinæ*).—Idiopathic separation of the retina from the underlying choroid is due to an accumulation of a serous fluid between these membranes (*serous detachment of the retina*).

Symptoms.—The student will observe, as he examines the various portions of the fundus with the ophthalmoscope (direct method), an alteration of refraction at the area of separation, the surface of the elevation thus produced being out of focus as compared with the rest of the eye-ground. Thus, if the general fundus is hyperopic, the detached portion will be more hyperopic, and require a stronger convex glass for the study of its surface; if it is highly myopic, a weaker concave glass, or, it may be, a convex lens.

The normal color of the fundus is lost as the detached retina is approached, which appears as a gray or bluish-gray membrane stretching forward into the vitreous, containing folds which give rise to a sheen. The intervening furrows present a greenish-gray reflex, and the whole oscillates with the movements of the eye if the underlying substance is fluid; if it is a solid, neither folds nor tremulousness are present. Rents in the detached retina, through which the choroid is visible, are often demonstrable.

The retinal vessels rise over the separated portion, first lose the light-streak, and finally appear as dark, tortuous cords. They apparently are of smaller size than normal, and when followed backward they pass out of focus at the edge of the detachment, which is usually sharply marked from the normal fundus; indeed, there may be a yellowish border and occasionally accumulated pigment. The amount of discoloration of the detached area depends upon whether the case is recent or not, and upon the character of the underlying substance. In the earlier stages transparency is not lost, and the gray color, previously described, may not be present.

The detachment, either *partial* or *complete*, may occupy any portion of the fundus, but commonly is found below, preceded, it may be, by a separation of the retina in the upper part of the eye-ground. These superior detachments may sag downward and bulge forward into the vitreous like a diminutive balloon. Sometimes the detachments are quite small, like a series of furrows, and at other times an almost circular circumscribed separation occurs. Finally, the subjective signs of detachment may be present without discoverable elevation of the retina, but over the area (which subsequently separates) there is complete loss of the light reflex from the retinal vessels (Loring). In these circumstances the visual field should be mapped under reduced illumination. At first, owing to the small quantity of subretinal fluid, the detachment may be *flat;* occasionally it remains so, but usually it increases in extent and elevation.

Unless the macular region is directly involved, vision is not obliterated, but there is always interference with sight. This may develop

suddenly. The field of vision is lost in an area corresponding to the detached retina, and the completely darkened portion is usually bordered by a zone of imperfect vision corresponding to an area of retina not yet separated, but elevated above its normal plane. If the retina is detached below, the upper portion of the visual field is obliterated; if above, the lower portion, and so on (Fig. 201). A retinal detachment just beginning may not be detected by a visual-field examination with a white test-object, but may be represented by a relative scotoma if the test-object is blue.

The patients are conscious of distortion of objects (metamorphopsia); of floating spots before the eyes, due to the frequent presence of vitreous opacities; of an appearance like a cloud, due to the scotoma produced by the separated area; and of phosphenes, although the last cannot be elicited by pressure on the eyeball over the separated area. Should a

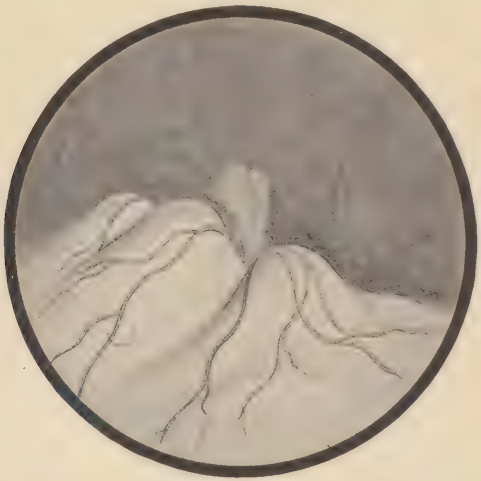


Fig. 200.—Detachment of lower half of retina, which has floated forward. Disk and upper half of fundus dimly seen.

patient with high myopia describe "attacks" of flashes of light, showers of sparks, or other types of photopsies, such symptoms are dangerously significant of impending retinal detachment.

Causes.—The causes of retinal separation are: High (malignant) myopia; traumatisms and effusions of blood, preceded usually by hemorrhages into the vitreous or retina. More men than women acquire simple detachment of the retina; myopic refraction most frequently is present, and the separation is more apt to occur in an eye in which the visual disturbance has rapidly developed. The condition often becomes apparent suddenly, especially after physical exertion; occasionally it develops gradually.

Retinal detachment is also caused by intra-ocular tumors (sarcoma of the choroid) or subretinal parasites (cysticercus), tumors and abscesses in the orbit, and diseased conditions of the eye, as retinitis,

cyclitis, iridocyclitis, etc. In iridocyclitis the detachment is often found only after removal of the shrunken globe, and is caused by contraction during organization of strands of connective tissue attached to the retina. Separation of the retina may be *congenital*, due to syphilis, and it has been observed in several members of the same family (*hereditary detachment of retina*—Pagenstecher). Subretinal hemorrhage —the blood coming either from the retina or choroid—may produce the so-called *hemorrhagic detachment of the retina*, for example, in the subjects of arteriosclerosis, but also in anemia and chlorosis. In *traumatic detachment* of the retina the eye is usually, but not necessarily, myopic; the separation may immediately follow the injury or be delayed for weeks or even months (Galezowski).

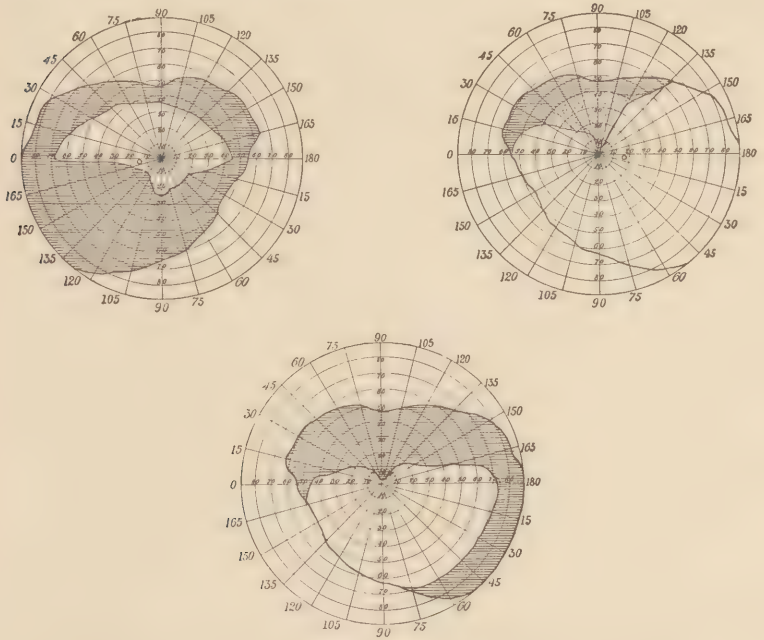


Fig. 201.—Various types of fields of vision in detachment of the retina.

Mechanism.—Leber and Nordenson hold that the first process is a fibrillar change in the vitreous, which shrinks and occasions traction (retraction theory). This ruptures the retina, and the fluid from the vitreous cavity passes beneath it through the opening. The primary cause of the pathologic alteration in the vitreous is believed to be disease of the choroid and ciliary body. Greeff, Elschnig, and others, however, doubt if retinal detachment is caused by shrinking of the vitreous; detachment of the vitreous they regard as an artefact due to the action of fluids used in hardening the eyeball. Raehlmann explains the detachment by a *diffusion theory*, the conditions being analogous to transudations in other parts of the body. In some instances retinal detachment must

be explained by the presence of exudation or hemorrhagic extravasation, and in axial myopia of high degree the "mechanical theory" is applicable—the separation of the retina being due to elongation of the eyeball, fluidity of the vitreous, and hyperemia of the choroid.

Diagnosis.—No difficulty arises in detecting a large detachment of the retina by attending to the symptoms already detailed. An extensive or complete detachment which floats far forward may be examined by oblique illumination. If the vitreous is full of opacities, a study of the field of vision is useful. If the substance underlying the detached portion is *fluid*, there are usually diminished tension of the eyeball and the appearance of furrows in the separated tissue, which trembles with the movements of the eye, symptoms which are absent if a *solid growth* has caused the separation. Important diagnostic signs are the loss of the light-reflex of the vessels, and their dark color over the area of separation. They can be seen to regain the light-reflex in passing over the normal retina. It is important to submit all eyes with detachment of the retina to the transillumination test (see page 363).

Prognosis.—This is very unfavorable, and many of the suggested means of treatment have proved unsatisfactory. In rare instances there is spontaneous reattachment of the retina.

Treatment.—This should include rest in the prone position, a pressure bandage, preferably elastic, and pilocarpin sweats, the pupil of the affected eye being dilated with atropin, and lymphagogue activity stimulated by the use of a 5 per cent. dionin solution. Internally, the various iodids or small, frequently repeated doses of salicylate of sodium may be administered.

Various forms of operative procedure have been attempted: sclerotomy and, recently, trephining the sclera, thus evacuating the subretinal fluid; evacuation of the subretinal fluid by puncture and aspiration, and drainage by means of a gold wire; electrolytic puncture; incision of the fibrous bands in the vitreous, followed by the injection of the vitreous humor of a rabbit (Deutschmann). Subconjunctival injections are valuable. For this purpose de Wecker employed a solution composed of $3\frac{1}{2}$ parts of gelatin with 100 parts of physiologic salt solution. Jocqs advocates injections of a saturated solution of salt in conjunction with scleral puncture, while Bourgeois recommends a 30 per cent. salt solution to which a few drops of a 5 per cent. solution of cocain are added, 1 c.c. of the fluid being injected. Subconjunctival injections of cyanid of mercury and of sodium chlorid are also advocated; they may be rendered painless by adding acoin to the solution. The author has secured a few favorable results with scleral puncture, followed by large (30 minims—1.9 c.c.) injections of physiologic salt solution, and has not found it necessary to increase the strength of the salt solution beyond 4 or 5 per cent. During the treatment the patient should remain in bed. Scleral cauterization, followed by subconjunctival saline injections, has been employed by Dor, and puncture of the eyeball with the galvanocautery has been advocated. With Deutsch-

mann's operation (bisection of the vitreous and retina, with or without the intravitreous injection of animal vitreous) the author has had no experience. Deutschmann reports good results; some surgeons who have investigated the subject are not in favor of the operation. If the detachment is due to a tumor, the eye should be enucleated. For further consideration of this subject, see page 619.

Hemorrhages in the Retina (*Apoplexy of the Retina*).—The appearances of retinal hemorrhage have been described in the general symptom-grouping, and as they occur with so-called hemorrhagic retinitis (see page 432).

Hemorrhages (unassociated with inflammation) may be in any of the layers of the retina, or, bursting through the limiting membrane, they may occupy the vitreous humor. By preference they are found along the course of the larger vessels; a favorite site is the macula. Hemor-

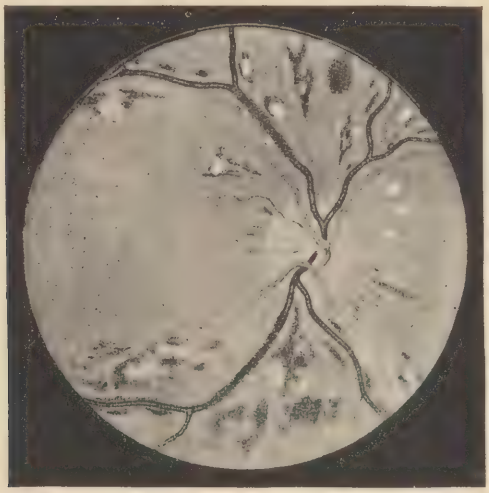


Fig. 202.—Retinal hemorrhages (de Wecker and Masselon).

rhages originating in the outer sheath of the optic nerve may appear at its margin and spread into the surrounding retina, although it does not follow that intrasheath hemorrhagic extravasations always follow this course.

Hemorrhages of large dimensions and drop-like form usually mean an extravasation between the internal limiting membrane of the retina and the hyaloid membrane of the vitreous, and they come from a retinal vessel. These *subhyaloid* or *preretinal hemorrhages* tend to occur at the yellow spot more than at other parts of the fundus. Occasionally they assume a wedge-, bottle-shaped, or almost circular appearance, or they may have, as in an eye recently examined by the author, a long, roll-like form, and overlie the sweep of the retinal vessel; often they are semilunar in shape. Except as the result of traumatism they are exceedingly scarce in young children, but Harms has reported one case in a

child of four and one-half years. According to J. Herbert Fisher, the hemorrhage detaches the internal limiting membrane from the retinal layers, which are not invaded, and occupies the space thus formed. It may break into the vitreous.

Causes.—Some of these have already been enumerated. The following résumé, based upon the classification of Dimmer, may be added:

(*a*) Hemorrhages caused by changes in the composition of the blood and the tissues of the blood-vessel walls: Pyemia, septicemia, ulcerating endocarditis; diseases of the liver, spleen, kidney, and atheroma of the vessels, and angiosclerosis of the retinal vessel; loss of blood (menorrhagia, hematemesis); anemia (simple and pernicious); hemophilia, purpura, and scurvy; diabetes and gout; tuberculosis; malaria and recurrent fever. In carcinoma of the stomach retinal hemorrhages and white spots may resemble those seen in pernicious anemia. To this manifestation the name *cachectic retinitis* is sometimes applied.

Fig. 203.—Subhyaloid hemorrhage.

(*b*) Hemorrhages caused by disturbances in the circulation: Hypertrophy of the heart and stenosis of the valves; thrombosis of the central vein of the retina, and embolism or thrombosis of the central artery; suffocation, compression of the carotid, and hemorrhages in the newborn, which are not infrequent; and the menstrual disturbances.

(*c*) Hemorrhages caused by sudden alterations of the intra-ocular tension—*e. g.*, after iridectomy in glaucoma—and by traumatisms. Among the latter may be classed retinal hemorrhages after large cutaneous burns, and those which have followed compression of the thorax and neck and fracture of the skull.

(*d*) Hemorrhages caused by certain toxic agents—*e. g.*, phosphorus, chlorate of potassium, serpent virus.

Prognosis.—This depends upon the extent and situation of the hemorrhages. They form an important prognostic guide of the disease which has caused them, and in elderly persons may be an indication of future hemorrhages into the brain. Glaucoma, detachment of the retina, and the formation of dense opacities in the vitreous humor may be complications.

Treatment.—All use of the eyes must be forbidden. Locally, a weak solution of sulphate of eserin may be employed, especially in elderly people. Internally, the medication must be governed by the probable cause. Frequently cardiac sedatives, moderate diaphoresis, and later, alteratives, such as iodid of potassium, iodid of sodium, syrup of hydriodic acid, and bichlorid of mercury, will be required. If the arterial tension is high, nitroglycerin should be administered.

Fig. 204.—Extensive retinal vessel disease; periarteritis and periphlebitis. Right eye.

Changes in the Retinal Vessels and Their Walls.—Certain changes in the retinal vessels due to *vasculitis* and *perivasculitis* are often seen. These are characterized by the appearance of white stripes along the vessels or, rather, the vessel walls become apparent by their conversion into whitish tissue, due probably to an infiltration of the adventitia with lymph-corpuscles. This may be so extensive that the entire set of vessels is converted into a series of branching white lines.

Such conditions may be due to various inflammatory diseases of the retina and optic nerve. Alterations in the retinal vessels are also caused by *chronic nephritis* and *general arteriosclerosis*, and present the following ophthalmoscopic appearances:

1. Alterations in the course and caliber of the retinal arteries, manifesting themselves as (*a*) undue tortuosity, which is not significant unless, to quote the words of Mr. Gunn, whose classification is followed, it is associated with other evidence of disease; (*b*) alterations in

the size and breadth of the retinal arteries, presenting, as it were, a beaded appearance.

2. Alterations in the reflections from, and the translucency of, the walls of the retinal arteries, manifesting themselves (*a*) in increased distinctness of the central light-streak on the retinal vessel and an unusually light color of the entire breadth of the artery ("silverwire arteries"); (*b*) loss of translucency, so that it is impossible to see, as it is in the normal state, through the artery an underlying vein at the point of crossing; (*c*) positive changes in the arterial walls, consisting of whitish stripes, indicating degeneration of the walls or infiltration of the perivascular lymph-sheaths (*perivasculitis*).

3. Alterations in the course and caliber of the veins, together with signs of mechanical pressure, manifesting themselves (*a*) in undue tortuosity, which, as in the case of the arteries, is not significant except in the presence of other disease; (*b*) alternate contractions and dilatations; (*c*) an impeded venous circulation where a diseased artery crosses it. The last is a sign of the utmost importance. Ordinarily, as an artery crosses the vein, as it may be seen by an examination of the normal eye-ground, there is no sign of pressure. If the walls of the artery are thickened by disease, it presses upon the vein, pushes it aside, or directly contracts its caliber, so that beyond the point of crossing there is an ampulliform dilatation. (*d*) Changes in the venous walls, precisely as they occur in the arteries, so that whitish stripes border the vessel, and are indications of degeneration in its walls. Often associated with this one may see varicosities. (See Plate V.)

4. Edema of the retina, manifesting itself (*a*) as a grayish opacity, which may be present in the immediate neighborhood of the papilla, or in spots over the eye-ground and along the course of the vessels, looking like a fine gray haze, or in little fluffy islands far out in the periphery.

5. Hemorrhages, manifesting themselves as linear extravasations along the course of the vessels, roundish infiltrations scattered over the fundus, or sometimes in a drop-like form. All these changes have been described by Raehlmann, Friedenwald, Hirschberg, the author, and many other observers, and were especially accurately recorded and classified by the late Mr. Marcus Gunn.

The *significance* of these lesions is of serious import. In addition to their relation to nephritis, they may be the forerunners of vascular sclerosis of the brain or indicate the presence of disease of the cerebral arteries. Their subjects are liable to hemorrhage in the brain and all its consequences. In *retinal angiosclerosis*, the disease is probably primarily in the intima, according to Coats, and in arteritis new tissue forms which encroaches on the lumen of the vessel, sometimes suggesting a hyaline change. According to this observer, irregularities of vessel caliber are due to endothelial proliferation, and silverwire arteries to fibrosis. Endothelial proliferation of the main artery depends upon a circulating toxin.

PLATE V.

Changes in the fundus in arteriosclerosis.

Angioid Streaks in the Retina (*Retinal Pigment Striæ*).—These occur as dark, reddish-brown, sometimes almost black striæ lying beneath the retinal vessels. They give the impression of a system of obliterated vessels, as in a case recorded by the author, but are caused, according to Ward Holden, by the metamorphosis of hemorrhages diffused in a linear manner through the deep layers of the retina. Lister thinks they represent newly formed vessels which have penetrated inflamed tissue, and along which pigment deposits and other exudations are arranged. W. Zentmayer is inclined to regard the streaks as pigmented vessels which are either of inflammatory or congenital origin. Coats suggests that the streaks arise along vessels, but that these vessels belong to the choroid rather than the retina.

Fig. 205.—Angioid streaks in the retina; large central semicircular hemorrhage.

Exudative Retinitis (*Retinitis Hæmorrhagica Externa; Massive Retinal Exudation* [Coats]).—In this form of retinal disease, particularly well described and studied by George Coats, the most conspicuous feature is a large, prominent yellowish-white circumscribed lesion, or smaller areas of yellow or white exudations lying beneath the retinal vessels. Coats has investigated several varieties of the affection, namely, those without gross vascular disease, those with extensive vascular changes, and those with arteriovenous communication. Recently he has removed the last-named variety from his classification of massive retinal exudation. Of insidious onset and slow progress, the disease most often attacks young persons (average age about nineteen), and is more common among males than females. The patients are

usually in good health (anemia may be present), and their clinical and family histories do not yield information as to the etiologic factor. In late stages of the disease detachment of the retina, cataract, iritis, and glaucoma may develop. The affection depends, as Coats has shown, upon hemorrhages in the interretinal layers. A slow organization takes place with formation of cicatricial tissue masses. At first the choroid remains free from pathologic alterations, but there are borderline cases of the disease characterized by involvement of the choroid as well as the retina in the form of thickening and round-cell infiltration. Exudative retinitis is probably the result of local vascular disease;

Fig. 206.—Massive retinal exudation. Left eye of a girl aged nineteen.

ophthalmoscopically it has most often been mistaken for tuberculous choroiditis.

Aneurysms.—Aneurysm of the central retinal artery is an extreme rarity. It has been seen as a spindle-shaped sac, pulsating synchronously with the heart. Miliary aneurysms, usually spindle shaped, have been noted in the small arterial twigs, and may be looked upon as significant of a similar condition of the vessels in other organs, especially the brain. The student should not mistake varicosities in the veins for aneurysms. Arteriovenous aneurysm of the retina has been described as the result of injury (Fuchs) and as a congenital condition (Leplat). E. von Hippel, however, doubts the applicability of the term, because he believes that in these cases arteriovenous aneurysm was lacking.

Angiomatosis of the Retina.—This rare disease has been observed by Fuchs (see preceding paragraph), Goldzieher, Darier, and a few

other observers, but its first accurate description is by von Hippel. According to him, the most prominent ophthalmoscopic appearances are red, spheric formations, with enormous dilatation and tortuosity of one or more arteries and the accompanying veins, both sets of vessels having the same color. About 30 cases are on record; tuberculosis is suggested as a cause. Anatomic examination reveals diseased retinal vessels, destruction of the nervous elements, proliferation of the glia, and organization of subretinal hemorrhage (von Hippel). According to Meller, primarily there is increase of the neuroglia with secondary vascular changes. The prognosis as to vision is fatal; blindness from secondary glaucoma results.

Obstruction of the Central Artery of the Retina, Including Embolism and Thrombosis.—Rarely an embolus lodges in the central artery of the retina; 5 cases on record, according to Coats, namely, his own and those of Harms, Schweigger, Manz, and Marple, were definitely embolic in nature. Usually the symptoms recorded in the following paragraphs are caused by thrombosis or by obliterating endarteritis.

Symptoms.—The main branches of the *artery* are thin, and can be followed only a short distance over the edge of the papilla into the retina, and there is a diminution in the number of ramifications. The *veins* are also contracted, and very often they present unequal distention. They may present ampulliform broadening, alternate contractions and swellings, and especially a contraction at the disk, succeeded by broadening in the periphery, where they assume almost their natural breadth. Pressure from before backward, so as to increase the intraocular tension, causes a regular current to flow through the vessels. This consists of broken cylinders of blood, separated by clear spaces, which move sluggishly along. In the veins, without such pressure, and, it may be, directly after the accident, an *intermittent blood-stream* is often visible. The appearance is not unlike that produced when air is allowed to mix with a fluid in a tube. Occasionally a few hemorrhages are seen along the course of the vessels.

The *papilla* assumes a pallid, grayish-white appearance, owing to the lack of blood in its capillaries. An *opacity in the retina* develops, grayish-white or fog-like in appearance, sometimes permitting the reddish tint of the normal eye-ground to shine through it, and sometimes being so opaque that it is quite milk-like in its density. This occurs especially in the neighborhood of the papilla and in the macular region, the space between the two often being free, although gradually the areas meet. The opacity comes on within a few hours after the accident, or may be delayed for a day or two. The author has watched it form within twenty minutes after sudden stoppage of the central retinal circulation. It is due, according to Coats, not to edema, but to an *ischemic necrosis*.

Characteristic of sudden obstruction of the arterial circulation is the formation in the macula lutea (corresponding to the position of the fovea) of a central red spot, which resembles a round hemorrhage in the midst of the milky-white area. It is known as the *cherry-red spot*

of the macula lutea, and is caused by the red color of the choroid appearing through the much-thinned retina, and changes in the pigment epithelium. As a rare phenomenon, at least in the dark-skinned races, the usual cherry-red spot has been replaced by a coal-black one. The spot appears at the same time with the opacity in the macula lutea. It is less likely to form where there is a stoppage of a branch of the retinal artery instead of one of the main trunks.

In the course of several weeks there is a gradual disappearance of the retinal opacity, the optic disk undergoes atrophy, and the retinal vessels are shrunken or even converted into white cords; if there have been hemorrhages, spots of degeneration appear at their positions, and

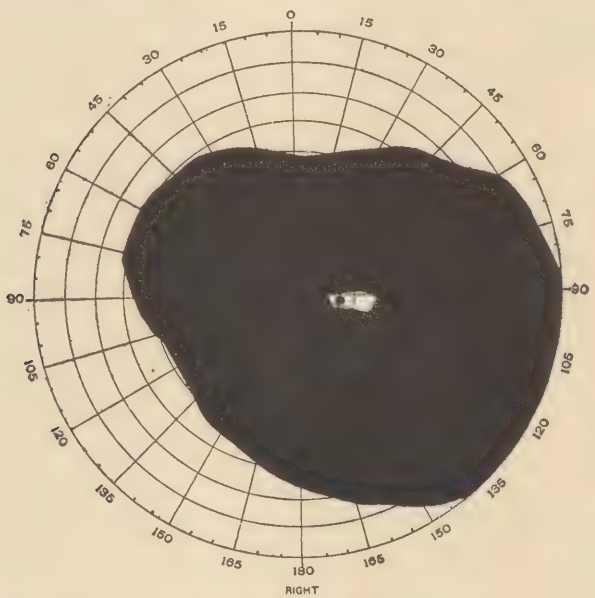


Fig. 207.—Small visual field representing area of functionating retina surrounding blind-spot.

not infrequently cholesterin crystals and pigment markings may be seen around the disk and in the macula lutea.

Instead of the *main trunk*, a *branch* may be obstructed, and this obstruction is sometimes visible to the ophthalmoscope as a yellowish body, but, more frequently, at one point of the artery there is a swelling, while beyond it the vessel is obliterated or its caliber is much reduced.

Vision is lost with characteristic suddenness. Usually preceding the blindness there is temporary obscuration of vision, or a little headache and giddiness, with flashes of light, representing a species of aura. Periods of temporary blindness lasting from a few minutes to one-half hour, during several years (in one of the author's cases twelve years), may precede the ultimate obstruction of the artery. In obstruction of a branch, on the other hand, there may be very good acuteness of vision.

The presence of a *cilioretinal vessel* may be the means of preserving vision. A rare condition is obstruction of a cilioretinal artery (Hirsch, Zentmayer, F. Krauss).

The *field of vision* varies according to the extent of the blocking of the circulation. In cases where the obstruction is complete, even light perception is absent. If only a branch has been occluded, that portion of the retina which receives its blood-supply from this source will be paralyzed, and the opposite area of the field will be darkened. The presence of a cilioretinal vessel permits, as a rule, an oval portion of the field of vision to remain in the neighborhood of the fixation-point, but, according to C. F. Clark, the evidence is not sufficient to warrant the conclusion that such a vessel is the means of preserving the integrity of the papillomacular region of the retina. Even if the main stem of the artery is obstructed, a small portion of the field may be preserved on the temporal side, corresponding to an area on the nasal side of the fovea in the region of the blind-spot. An uncommon effect is a central scotoma, which may be due to obstruction of the macular arteries; the scotoma may also be paracentral.

The intra-ocular tension is sometimes raised, sometimes lowered, and sometimes unaffected. The pupil may be large and irresponsive to light if the case is one of complete stoppage of the central artery.[1]

Causes.—The most frequent causes of obstruction of the central artery of the retina are valvular disease of the heart, especially if complicated by a fresh endocarditis, and general arterial sclerosis, aneurysm of the aorta or of the carotid, Bright's disease and pregnancy; in a few instances it has been noted with chorea. Thrombotic obstruction depends upon endarterial changes, alteration of the composition of the blood and its coagulability. Stoppage of the central artery may occur at almost any age of life, and has been recorded from the fifteenth to the eightieth year. The accident usually is unilateral, simultaneous obstruction of the central artery of each eye being very rare; occasionally both eyes are affected, with a definite interval between the attacks.[2] Blindness with ophthalmoscopic appearances exactly similar to those caused by obstruction of the central artery has followed subcutaneous injections of paraffin in the nasal region and of bismuth paste into the accessory sinuses.

Diagnosis.—The ophthalmoscopic picture just detailed indicates that a block in the central retinal circulation has occurred, due usually to one or other of the causes already named. Similar appearances have been ascribed to hemorrhage into the sheath of the optic nerve, to spasm of the muscular wall of the central artery, and to thrombosis of the central vein so situated that it presses upon and occludes the lumen of the artery lying beside it. Schweigger taught that emptiness of the arteries was an important sign of true embolism. Some cases of

[1] The symptoms which have been described refer to typical cases; a variety of exceptions occurs.

[2] In a certain number of cases, although all the ordinary ophthalmoscopic appearances of embolism of the central artery of the retina have been present, it has been impossible to assign a cause.

obstruction of the central retinal circulation appear to be due to collapse of the arterial walls, so that they come in contact (Hoppe). In these circumstances recovery may occur spontaneously or be brought about by treatment. While it is true that "there is at present no proof that obstruction may be caused by spasm apart from endarteritis" (Coats), the effect of an apparent spasm from the clinical standpoint must be conceded. The author has examined Harbridge's patient, and watched complete collapse of the retinal arteries, followed in four minutes by the restoration of their caliber. *Intermittent closing of the retinal arteries*, apparently due to vessel cramp, has been many times recorded; for example, with angiosclerosis, Raynaud's disease, epilepsy, and migraine (see also page 422).

Prognosis.—This is exceedingly unfavorable, and in most instances blindness is the result. Even when temporary improvement occurs, subsequent atrophy of the nerve is likely to ensue. In obstruction of a branch the prognosis is more favorable, and, as has been stated, normal central vision may be present. The presence of a cilioretinal vessel improves the prognosis. According to Swanzy, in cases of bilateral obstruction, unless vision is lost simultaneously, a fair amount of vision may be restored. Glaucoma may follow stoppage of the central retinal artery.

Treatment.—This does not often prove of much avail. In the hope of restoring the circulation by reducing the intra-ocular tension, sclerotomy, iridectomy, and repeated paracentesis of the anterior chamber have been practised, but without success.

Massage of the eyeball has been recommended, and in some cases has been followed by good results. It should be given a faithful trial; the author can confirm its value. With the massage, inhalations of nitrite of amyl may be given (Gifford).

Thrombosis of the Central Vein.—This has been observed a number of times as the result of a phlebitis, and also in patients with cardiac disease, arteriosclerosis, nephritis, and diabetes.

In some instances the appearances have been closely similar to those of embolism; in others they have assumed an inflammatory character similar to that described under Hemorrhagic Retinitis, of which it may be a cause (see page 432). Several grades of this condition have been recorded. If the ophthalmoscope reveals tortuosity of the vessels, engorgement of veins, and normal or contracted caliber of the arteries, venous pulse, and interrupted venous circulation and extensive retinal hemorrhages, thrombosis of the central vein may be suspected (Angelucci). There may be complete obscuration of the disk, which is hidden by infiltrated retina, and surrounded by large flame-shaped and sometimes sheet-like hemorrhages, which extend widely over the fundus. Instead of the main trunk, one of its branches may be thrombosed, and the ophthalmoscopic appearances confined to the area which it drains (Fig. 208). According to conditions various visual fixed alterations are demonstrable; a central scotoma may be present.

Prognosis.—If the thrombosis is in the central vein, hope of res-

toration of vision practically must be abandoned; in obstruction of a branch the prognosis is more favorable. A *secondary glaucoma*, sometimes developing acutely, is not an uncommon complication, due to obstruction of the filtering area by an albuminous exudate. *Iritis* with the formation of fibrovascular membrane has been observed.

Treatment.—This depends upon the general condition. Other things being equal, iodids and syrup of hydriodic acid may be administered. Diaphoresis may be tried; mydriatics should not be used lest they cause glaucoma.

Traumatisms of the Retina.—Under this general term may be included traumatic anesthesia, traumatic amblyopia, traumatic perforations of the macula lutea, detachment, and rupture. There are no characteristic symptoms common to all varieties, but pain and dis-

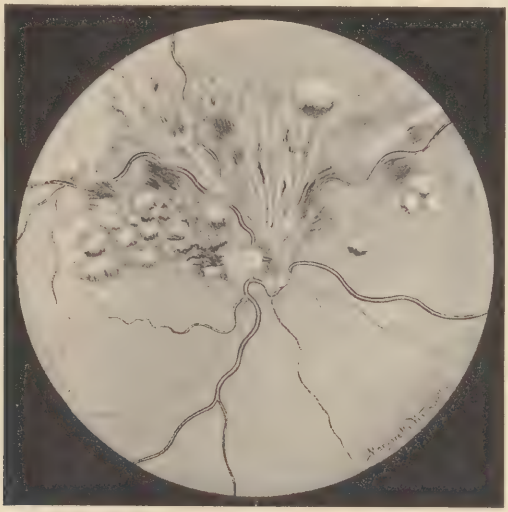


Fig. 208.—Thrombosis of a retinal vein.

turbance of vision, in part due to the direct injury and in part to a transient astigmatism, are likely to be present.

1. *Traumatic anesthesia of the retina* is the name proposed by Leber to describe effects of a blow upon the eye without discoverable ophthalmoscopic changes, but with considerable defect in vision and contraction of the visual field—results, moreover, which may remain unchanged for a long time, or, indeed, never entirely pass away.

The *treatment* is rest and the use of strychnin internally, or by hypodermic medication.

2. *Traumatic amblyopia* (*commotio retinæ; edema of the retina*) is a condition also arising from an injury, especially a blow from a ball, cork, or similar body, and is attended by the following symptoms: Hyperemia of the globe marking the position of contact of the missile; clear media; and gray opalescence of the retina, especially in the macular region, but also around the papilla, which may be somewhat hyperemic. If

the retina under the point of contact is visible, this also may exhibit the white infiltration. In addition, several pale-yellowish spots, and, occasionally, small hemorrhages may be present. The vessels are unchanged, or, in some instances, are contracted (arteries) or distended (veins) and pass *over* the gray area. A central scotoma may exist.

An interesting complication is the development of a transitory astigmatism, which helps to reduce the visual acuteness.

The gray infiltration forms quickly and is also absorbed with rapidity, usually having subsided at the end of two or three days, although the visual defect may last for longer periods. Decided retinochoroiditis, the result of concussion, may occur, and this fact should be remembered

Fig. 209.—Hole in macula and rupture of choroid after a blow on eye (patient in the University Hospital).

in investigating old cases of choroidal disease presenting themselves with meager history. According to Fuchs, changes in the macula after contusion may be due to inflammatory edema as the result of a low-grade inflammation of the ciliary region.

The *treatment* consists in keeping the pupil dilated with atropin and covering the injured eye with a shade or dark glass, all use of the uninjured organ being forbidden.

3. *Traumatic Perforations of the Macula Lutea.*—Haab has called attention to the fact that a contusion or concussion injury of the eye may cause a round hole in the macula about half the size of the surface of the optic disk, surrounded by a gray ring. The bottom of the hole is of reddish color, with a stippling of white and red. F. M. Ogilvie, who

calls the affection "holes in the macula," points out that these perforations are the immediate and direct result of the injury. In his observations these were represented by areas depressed below the level of the surrounding retina, of a deep-red color, and sharply margined by clean-cut edges. In some cases the retina is detached; in others it is not. A central scotoma may exist. Other signs of injury may be present in the eye-ground—for example, rupture of the choroid—as in an eye observed by the author. This "hole" in the macula is produced by an edema of the retina at the posterior pole (Coats).

4. *Detachment of the retina after injury* has been mentioned (see page 448).

5. *Rupture of the Retina.*—Rupture, uncomplicated by choroidal fissure the result of injury, is a rare accident, and might be recognized by observing the frayed edges of the tear and seeing the exposed choroidal tissue. Long describes such an occurrence following a fall upon the back of the head.

Purtscher has described a fundus lesion after fracture and traumatisms of the skull, to which he gives the name *angiopathica traumatica retinæ*. The ophthalmoscopic picture consists in the presence of shining white patches, chiefly associated with the veins; hemorrhages may also occur. These patches are supposed to be due to cerebrospinal fluid which has been forced into the retinal perivascular lymph-spaces. To this condition Körber applies the term *lymphorrhagia of the fundus.*

Retinal Changes from the Effect of Sunlight (*Solar Retinitis*) **and Electric Light** (*Electric Retinitis*).—It has been experimentally proved that retinal changes can be produced in animals' eyes by concentrating upon them the rays of the sun. Clinically, analogous disturbances have been found in the human retina after exposure to intense light, most frequently in those who, with unprotected eyes, have watched an eclipse of the sun (*eclipse blindness*). Similar conditions are caused by intense electric light, especially among those engaged in electric welding.

The **symptoms** are: Persistence of an after-image or, later, a dark spot in the field of vision (positive scotoma); distortion of objects, and evidences of slight retinitis or retinochoroiditis in the macular region. Thus, there may be a maroon-colored area with a central gray patch, and numerous faintly marked yellowish-white dots. A cherry-red spot has been detected by Ischreyt and a definite "hole in the macula" has been described.

Decided improvement is not infrequent, but complete recovery is exceptional (Mackay); hence prognosis must be guarded. The central scotoma may be permanent (Duane); ring-shaped scotoma has also been observed. Degeneration of the papillomacular bundle may occur (E. T. Collins).

The **treatment** is that suited to retinochoroiditis. The preventive treatment consists in wearing suitable colored glasses—yellow glass or a combination of blue and red, or, as in Sheffield, several layers of ruby glass.

Glioma of the Retina.—This is a malignant growth of the retina, and is a soft, vascular tumor, made up of small round, deeply staining cells, many of them containing long protoplasmic processes. They form thick mantles of well-preserved cells around the thickened blood-vessels, the cells between the mantles staining poorly and undergoing calcareous degeneration. In many of these neoplasms peculiar *rosettes* have been described by Flexner, Wintersteiner, and others, which are composed of elements resembling the rod and cone visual cells, and for these growths the name *neuro-epithelioma* has been suggested. Alt thinks the rosette formation is due to the growing of tumor-cells around a tissue enclosure, and not to rudimentary rods and cones, and Ginsberg believes they correspond to cells of the rudimentary retina, which are not differentiated into spongioblasts and neuroblasts. Glioma usually arises from the inner retinal layers; less frequently from the outer retinal layer. According to Leber, it may develop from various layers not only in different eyes, but in the same eye (Parsons). Exactly how glioma originates is not certainly known, but probably in fetal retinal cells.

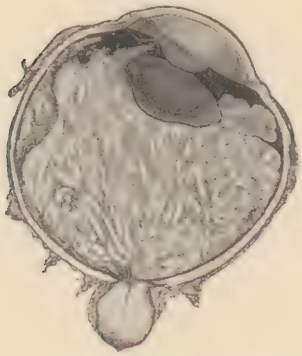


Fig. 210.—Glioma of retina (patient in the University Hospital).

According to the direction which the growth takes, it has been described by systematic writers as *glioma endophytum* and *glioma exophytum*. In the former the vitreous chamber is occupied by the growth; in the latter, it lies between the retina and choroid.

The tumor is usually of a light-gray or grayish-red color. It is subject to various degenerative changes—fatty, cheesy, and calcareous—and tends, on the one hand, to invade the orbit, involve the optic nerve, and travel by the way of its sheath to the brain, and, on the other, to pass forward, bursting through the sclerotic and cornea. Recurrence *in loco* after extirpation may occur, and metastases, especially in the cranial and facial bones and the brain, may take place. They also occur, according to F. M. Wilson and E. S. Thomson, in the lymph-glands, parotid, liver, ovaries, kidneys, spleen, lungs, and spine. Glioma may cause changes in and invade tissues of the eye other than the retina: the vitreous, choroid, iris, ciliary body, and anterior chamber.

Like sarcoma of the choroid, glioma passes through several stages. In the first, there are no signs of irritation, the media are clear, the pupil is dilated, and often the growth produces a whitish reflection which has given rise to the designation *amaurotic cat's eye*. As the disease progresses symptoms of irritation and increase in the size and tension of the globe become manifest, and the process begins to involve the optic nerve. Finally, the tumor bursts from its bounds, perforates the globe at its corneoscleral junction, grows rapidly, involving the orbit

and neighboring temporal regions, and presents a huge vascular mass, to which, in former times, the name *fungus hæmatodes* was applied.

Glioma of the retina is probably always congenital. It may apparently occur as late as the eleventh year, but in such circumstances it is probable that the growth has been present, but has remained quiescent. It is not a common affection. Hereditary predisposition has not been established. Several members of the same family may be affected. One or both eyes may be involved.

Diagnosis.—The following conditions, according to E. T. Collins, may be mistaken for glioma: Persistence of the posterior part of the

Fig. 211.—Recurrence of glioma, forming the so-called fungus hæmatodes (from a patient in the Philadelphia General Hospital under the care of Dr. Hearn. Photograph by Dr. Pfahler).

fetal fibrovascular sheath of the lens; masses of tubercle in the choroid; inflammatory or purulent effusion into the vitreous following retinitis or cyclitis, usually with detachment of the retina (see also Pseudoglioma, page 414). Circinate retinitis (white degeneration of the retina), according to de Wecker, has been mistaken for glioma. The author and E. A. Shumway have recorded a case of detachment of the retina with extensive dropsical degeneration of the rod and cone visual cells which exactly simulated glioma. In glioma the anterior chamber is uniformly shallow; in inflammatory exudations into the vitreous the chamber is deepened at its periphery (retraction of iris) and shallow at its center (bulging of pupillary border). Synechiæ are occasionally present in

glioma. Tension is usually increased in glioma, but may be minus; rarely the tension is elevated in pseudoglioma. In case of doubt the eye should be enucleated.

Sarcoma of the choroid is differentiated from glioma by the fact that the former usually occurs at a later period of life, and that in the earlier stages of each affection the ophthalmoscopic findings are different. In glioma the tumor is seen to *involve* the retinal structure, which does not, as in sarcoma, merely act as a covering to the growth. Unlike sarcoma, glioma is never pigmented.

Prognosis.—This is unfavorable, and if the disease has involved the optic nerve or bursts from its bounds, it is fatal. Although so-called spontaneous cure has been observed (Lindenfield) as an almost invariable rule, unmolested glioma causes death. Numbers of recoveries after proper enucleation are on record, and an opinion must be based on the extent of the disease, the condition of the optic nerve being the most important element in the prognosis. According to Hirschberg, a favorable prognosis may be given if the tumor has not passed the limits of the retina, and if the time elapsing since the first appearance of the growth has not exceeded ten weeks. Recurrence is rare after three years of immunity. In a number of fatal cases which have been analyzed (Lawford, Collins) the optic nerve was unaffected in only four. In unfavorable circumstances recurrence in the orbit occurs, with extension to the brain, and, more rarely, metastasis to a distant organ.

Treatment.—Thorough enucleation, with division of the optic nerve as far back as possible, is the only treatment. Any suspicious tissue in the orbit is to be sacrificed. In several instances both eyes have been removed, and recovery after such procedure has been recorded—for example, by Simeon Snell.

Subretinal Cysticercus.—This, like the presence of the same parasite in the vitreous, is exceedingly uncommon in this country.

Cysts of the Retina.—Small cysts or cystic spaces due, probably, to arteriosclerosis of the retinal capillaries are not infrequently found in the eyes of old persons just behind the ora serrata (E. T. Collins). Cysts are also found in the detached retinas of blind eyes, where they may be multiple. They may commence in the degenerated retina, or be due to the adhesion of folds of this membrane (Parsons). Sometimes they are visible with the ophthalmoscope. In a patient under the author's care a large retinal cyst springs from the upper part of an inferior detachment of the retina, leans over and partially obscures the disk. G. S. Derby describes a similar cyst.

Symmetric Changes at the Macula Lutea in Infancy.—This disease, which occurs almost always in children of Hebrew parentage and begins from the third to the sixth month of life, is also known as "amaurotic family idiocy" (a name given by B. Sachs); it was first described by Waren Tay, and consists of a grayish-white zone about the size of the papilla in each macular region, with a brownish or cherry-red spot in the center, closely resembling the appearances seen in obstruction of the central artery. At first the remainder of the fundus is normal, but

later the optic disks undergo atrophy. Kingdon has thus summarized the general clinical signs: muscular enfeeblement, apathy, mental weakness, and gradual loss of sight. Death occurs in from one to two years. The autopsies show a change in the pyramid cells of the cortex and degeneration of the cord. According to Sachs, this is an arrest of development. Ward Holden, by Nissl's method, has shown that there is degeneration of the retinal ganglion cells, and his results have been confirmed by Shumway and others. Verhoeff attributes the dark spot in the macula to contrast; no edema or hole was found in this region. The ocular conditions of this disease are merely, as Sachs insists, one symptom of a family affection. Syphilis is not a cause of the disease.

Fig. 212.—Changes at the macula lutea in amaurotic family idiocy (from a patient in the University Hospital).

Family Cerebral Degeneration with Macular Changes.—This affection, first described by R. D. Batten and named by Oatman "maculocerebral degeneration" (familial), presents itself in two forms: (*a*) The maculocerebral type, both retina and brain being affected, and (*b*) the macular type, the lesions being confined to the retina. It develops at a later age than family amaurotic idiocy, and is not race selective. In the first type the disease begins usually about the sixth year of life and is characterized by macular pigmentation, progressive blindness, progressive paralysis, and dementia (Batten and Mayou). In the second type the period of development is delayed and begins about the thirteenth year, and the central nervous system is not affected. Such cases have been described by Stargardt as "progressive family

degeneration in the macular region." Brown Pusey's investigations of this type of the disease suggest to him it may be an expression of early arteriosclerosis. These and allied lesions have been discussed by Gifford under the title of "Juvenile Types of Amaurotic Family Idiocy."

Macular Atrophy of the Retina.—Various types of central or macular retinochoroiditis have already been described, and it is not uncommon to find in the eyes of old persons, in the macular regions, areas of yellow-white spots interspersed with pigment dots and small hemorrhages, or irregular areas of erosion which may go on to atrophy of the elements and pigment heaping. Haab, however, has called attention to a pure retinal senile affection consisting of yellowish-red or whitish, or else darkly pigmented spots, the rest of the eye-ground being normal, and Harms, by microscopic investigation, demonstrated that the lesions depend upon an atrophy and disappearance of the involved tissues, affecting chiefly the neuro-epithelial layer. The pigment epithelium is much altered, but the choroid is practically not affected. Kuhnt, Haab, and the author have described a senile macular affection (retinitis atrophicans centralis), probably belonging to this class, which in all particulars in its ophthalmoscopic appearances resembles the lesion known as traumatic perforation, or "hole" of the macula (see page 462). It may be caused by retinal vascular disease, and a similar appearance may arise as the result of a non-traumatic iridocyclitis or from a toxin. Indeed, Fuchs has shown that hole-formation in the macula may occur in a variety of conditions; for example, iridochoroiditis, neuroretinitis, retinal detachment, and retinitis pigmentosa.

These macular changes do not respond to treatment.

CHAPTER XVI

DISEASES OF THE OPTIC NERVE

Congenital Anomalies.—Opaque or Medullated Nerve-fibers.—In the normal eye the fibers of the optic nerve cease to be invested with a medullary sheath at the lamina cribrosa, and consequently the axis-cylinders, which are distributed to the retina, are transparent. As an anomalous condition, sometimes bilateral, but more frequently only in one eye, the medullary sheaths reappear at the upper or lower margin of the disk as a dull or glistening bluish-white patch, which extends for a variable distance out into the retina, and ends in a somewhat feathery or fan-shaped margin. Usually the retinal vessels are hidden by the patch, but reappear again on its distal side.

This plaque may be a single one above or below, or it may appear both above and below the disk, more rarely on the nasal side, and very exceptionally upon the temporal margin. The size varies from a small expansion to a huge sweep of white tissue, continuous above and below with margins of disk, and taking somewhat the general direction of the vessels, which are wholly or in part concealed. Opaque nerve-fibers of the retina at a considerable distance from the disk have been recorded by Randall, Nettleship, Arnold Lawson, and other observers.

This condition produces no change in vision except an increase in the size of the normal blind-spot, and should not be mistaken by the beginner for pathologic lesions—for example, an atrophy of the retina and choroid, or a bank of fatty degeneration as it occurs in retinitis albuminurica.

Coloboma of the Sheath of the Optic Nerve.—This congenital anomaly is characterized by an apparent augmentation of the surface of the disk and an excavation of the papilla backward and downward. The periphery is usually bounded by pigment massing. There is an unequal division of the retinal vessels, which are first seen as they bend over the margin of the excavation. It is a rare anomaly, and has been mistaken for posterior staphyloma. It depends upon imperfect closure of the fetal fissure.

Irregularities in the Disk.—Instead of its usual round or oval shape, the disk may be markedly irregular in outline, one side being occasionally at an apparently lower level than the other, or it may present a gibbous appearance. *Congenital pigmentation of the optic nerve-fibers,* most intense in the position of the physiologic excavation, has occasionally been described; the pigment may exist as a small spot or cover an area one-fifth of the size of the disk.

When the nerve-head fails to fit the choroidal aperture accurately, a space is sometimes formed, usually crescentic, known as a "cone"

or "conus" (Loring). This generally is seen at the outer side of the papilla, but also inward, below, and very rarely above (see also page 131). It should not be confused with the cases of atrophy of the choroid seen in myopic eyes, to which the name *posterior staphyloma* is given (see page 132), nor with the crescents of choroiditis seen in astigmatic and stretching eyes, in which the scleral ring broadens out into a semi-atrophic area of disturbed choroid, usually bounded by an irregular pigment line, and most commonly developed at the temporal side of the disk. *Congenital inferior crescent* or *conus* is caused, according to Elschnig, by a pulling away of the choroid from the disk margin. The affected eyes are usually astigmatic.

Fig. 213.—Spurious optic neuritis.

Shreds of Tissue on the Disk.—These appear as glistening white patches of tissue, sometimes almost transparent, at other times thicker and more opaque, either completely or partially hiding the vessels (De Beck). Occasionally there is a white membrane more or less completely covering the disk.

Such appearances probably represent remains of the hyaloid artery or of its adventitious coat.

Hyperemia of the Nerve-head (*Congestion of the Disk*).—The color of the intra-ocular end of the optic nerve varies considerably, and it is not accurate to describe a nerve-head as congested if it simply is redder than usual.

As Gowers pointed out, the term *simple congestion* is applicable when the papilla presents a dull red or brick-dust hue, which shades almost imperceptibly, through a blurred margin, into the general red color of the fundus; when it is more marked in one eye than in the other, the latter serving as a picture for comparison; when at some antecedent examination the same optic disk has presented a more natural color; and when its borders are obscured, but not hidden.

In other circumstances—and the appearance is a frequent one—the surface of the nerve is covered by a semitransparent or edematous layer, is unduly injected, and its margins, especially the nasal ones, are veiled by striations composed of fine grayish lines and minute capillaries ordinarily not visible. The perivascular lymph-sheaths at the same time are unduly prominent in the form of white lines along the central vessels, especially the veins. This appearance has received the name "hyperopic disk," and has also been called "spurious optic neuritis" (Spicer) and "pseudoneuritis" (Stephenson). (See Fig. 213.)

Causes.—(*a*) Refractive error, especially hyperopia and hyperopic astigmatism. (*b*) Prolonged exposure to glare and heat. (*c*) Certain toxic agents presently to be described, and inflammation of the iris, usually of the syphilitic type. (*d*) Certain disorders of the brain, especially various types of chronic insanity. Focal brain lesion—for example, cerebral embolism—may be associated with hyperemia of the nerve-head.

Treatment.—This depends entirely upon the cause. Refractive error should be corrected if this is the apparent origin of the trouble. Constitutional measures will be required if there is reason to believe that some general cause is at work.

Anemia of the Nerve-head.—This is not a disease peculiar to the optic nerve, but, like retinal anemia, occurs as part of a general anemia, or because of obstruction to the central vessels—for example, in embolism.

It is most difficult to interpret the significance of pallor of the papilla. Usually it will require more than mere inspection to decide whether or not a pallid disk is pathologic.

1. **Intra-ocular Optic Nerve Inflammation and Edema.**—For convenience these conditions may be described as (*a*) *intra-ocular optic neuritis*, or *peripheral optic neuritis*, and (*b*) *engorgement-edema of the papilla*, or *choked disk*.

If the lesions consist of a hyperemia and a moderate swelling of the nerve-head and no unusual overfilling of the veins, and of an exudation which produces discoloration and opacity of the papilla, so that its margins and surface are obscured or hidden, and the whole process is not strictly limited to the disk, but passes into the retina immediately surrounding it, *descending neuritis* is often employed as a descriptive term.

If the lesions are chiefly confined to the nerve-head itself, and there are great swelling and engorgement, suggesting mechanical compression, marked distention of the retinal veins, and hemorrhages in and

near the edematous papilla, the term *choked disk*, or *papilledema* (Elschnig, Parsons) is used to describe the condition.

If the retina is extensively involved, with hemorrhages along the vessels, spots of degeneration, sometimes collected in a star-shaped figure analogous to that seen in renal retinitis, the term *neuroretinitis* is frequently utilized.

To avoid this confusion of names, Leber proposed the general term *papillitis*, and if it is understood to refer only to those types evidently of inflammatory origin, the word is suitable.

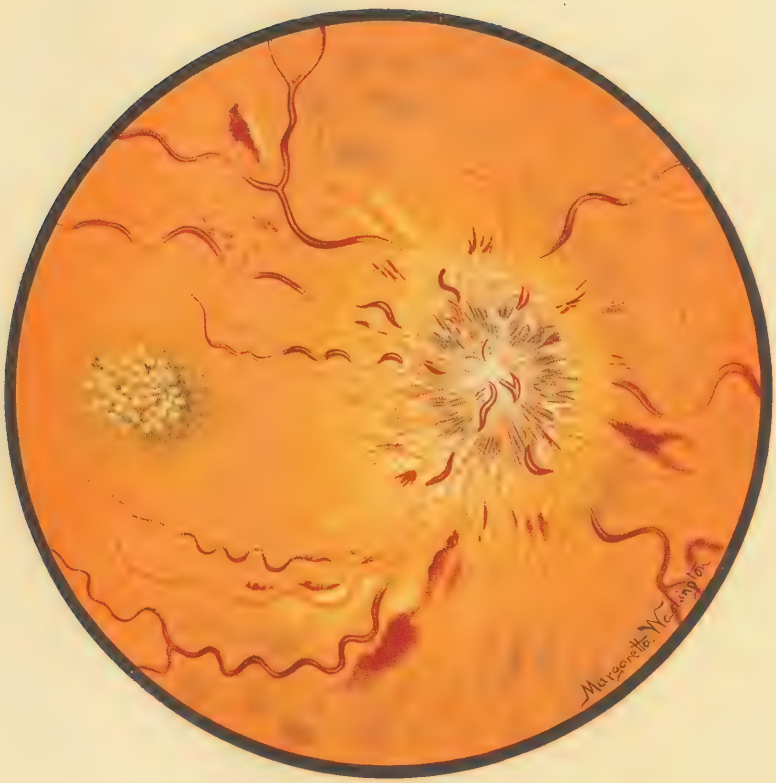
Symptoms.—In general terms, the symptoms which follow belong to the conditions now under consideration, but vary in their intensity or

Fig. 214.—Ophthalmoscopic picture of papillitis and semidiagrammatic representation of a longitudinal section of the nerve-head.

elaboration chiefly in so far as the swelling of the papilla is concerned, according as neuritis (optic neuritis) or engorgement-edema (choked disk, papilledema) is present.

1. *Changes in the Nerve-head.*—(*a*) There are increased redness of the disk and obscuration of its borders, followed by swelling of the papilla, loss of the light-spot, and complete hiding of the margins, the center usually remaining more red than the periphery, which has a grayish tint and shades gradually into the surrounding retina. The swelling may increase, assume a mound shape of mixed grayish color, and finally the form of the disk is lost, and its position can be inferred

PLATE VI.



The fundus of the right eye of a patient with tumor of the brain, showing choked disc.

only by the convergence of the vessels. The height of this swelling is measurable by the table given on page 107 and by the parallactic test. White spots and patches are often seen in the elevation sometimes covering the retinal vessel.

2. *Changes in the Vessels.*—The *arteries*, smaller than normal, pursue a moderately straight course and are difficult of recognition, being partly concealed by the swelling. Occasionally spontaneous pulsation is visible. The *veins* are dark in color, distended and tortuous, and pass along the slope of the elevation, often dipping into the infiltrated tissue. The light-streak is not lost, at least not where the vessel is clearly visible. The tortuosity of the vessels is sometimes remarkable, and has been compared to the writhing snakes in the *Medusa-head.* The point of emergence and convergence of the vessels may be hidden by the infiltration, so that the center of the swelling seems somewhat destitute of vessels. In some instances thickening of the adventitia of the vessels gives rise to the appearance of white lines along their sides.

3. *Hemorrhages.*—In many cases hemorrhages are found upon the swollen papilla or in its immediate neighborhood. They are in the form of narrow, flame-shaped extravasations if they lie in the fiber-layer, but may also assume other shapes if situated in a deeper plane. The number varies from a single hemorrhage to so many that the swollen nerve-head assumes a *hemorrhagic* form, or the surrounding retina may be freely occupied by elongated or other shaped patches of blood. Usually optic neuritis and choked disk are bilateral, although it is not uncommon to find the process more advanced in one eye than the other. Occasionally the condition is confined to one eye and the other remains unaffected.

In addition to the ophthalmoscopic changes just detailed, the following points deserve notice:

1. *Vision* in optic neuritis and in choked disk may be defective or it may be entirely unaffected; hence the mere presence of good central sight should never be considered cause to omit ophthalmoscopic examination. Usually the vision of one eye is more affected than its fellow. Impairment of sight may come on rapidly or slowly. Occasionally vision is lost with great suddenness, but this is rare. Photometric examination will usually reveal disturbance of the *light-sense* (see page 66).

2. The *field of vision* presents for consideration its periphery, which may at first be unaffected and later show irregular and concentric contraction; the increase in the size of the normal blind-spot, which becomes correspondingly great in comparison with the amount of swelling; the formation of an abnormal blind-spot or scotoma due to involvement of the axial fibers and occasionally simulating the Bjerrum type (V. Szily); the absence of half of the visual field (hemianopsia) if the intracranial mischief which may have been the cause of the neuritis or choked disk is so situated as to produce this phenomenon; and finally, defective color-perception, which may exist when there is no change in central vision and no limitation of the form-field. Cushing and Bordley have described reversal of the color lines as it

occurs in hysteria in association with increased intracranial tension, with and without choked disk, as well as blue-blind areas, which disappeared after the restoration of intracranial tension to normal by operation. Although reversal of the color lines in these circumstances undoubtedly is frequently demonstrable, it must not be regarded as a safe indication of the existence of increased intracranial tension.

3. *External Appearances.*—There are no changes in the exterior of the eye indicative of swelling or inflammation in the nerve-head. There are no characteristic *pupillary* phenomena. The pupil may be moderately dilated, but, as Kampherstein has shown, in the majority of cases of choked disk its reaction is normal. If blindness is complete, the iris usually is immobile. Normal reaction, however, has been noted even in the presence of complete blindness (Kampherstein).

Diagnosis.—The diagnosis of optic neuritis and of choked disk depends upon a direct ophthalmoscopic examination of the inflamed or edematous disk. The method of determining the height of the elevation has been explained.

The student should not mistake the slightly prominent disks that are occasionally seen in hyperopia for beginning papillitis or papilledema. There may be a superficial neuritis in hyperopia, and in these circumstances it is difficult at times to decide whether the disk has become edematous or inflamed under the influence of an intracranial or general disease, or whether it is congested as the result of eye-strain. If the condition is due to intracranial disease the disk edges are more blurred than in pseudoneuritis, the physiologic pit is contracted or filled in, the veins are darker and usually more tortuous, and a careful examination of the size and shape of the blind-spot, of the light-sense, and of the visual field should, in most instances, establish the diagnosis. The average swelling of papilledema in cerebral tumors is, according to the author's and Holloway's measurement, 4.57 D. It varies from 3 to 9 or 10 D.

The course of the disease is a variable one. Occasionally swelling of the intra-ocular end of the nerve will come on with great rapidity; in other instances it is slow in its course and lasts for months and even years, with progressive failure of vision. While in a certain sense the various stages into which systematic writers have divided choked disk (papilledema) and optic neuritis are artificial, they are convenient for descriptive purposes. The following, referring especially to choked disk, are those, somewhat modified, which were recorded by Marcus Gunn:

1. Increased redness of the disk, with blurring of its upper and lower margins, with a gradual progression of the blurring to the nasal edges, while the temporal margin is still visible, represents the first stage.

2. Increased edema of the nerve-head, beginning filling in of the physiologic pit, involvement of the temporal margin of the disk, with a tendency of the edema to spread into the surrounding retinal area, and uneven distention and darkening of the retinal veins represent the second stage.

3. Decided increase of edema, elevation and size of the nerve-head, with vascular striation of the swollen tissue and striæ of edema in the form of lines in the swollen retina between the disk and macula, marked distention of the retinal veins and retinal hemorrhages represent the third stage.

4. Increase in the prominence of the disk, which assumes a mound shape and begins to lose its reddish color and juicy appearance and to become opaque, exudation in and on the swollen disk and surrounding retina, elaboration of the retinal hemorrhages in size and number represent the fourth stage.

5. Decided subsidence of the vascularity of the papilledema and increasing pallor, with or without sinking of its prominence, apparently contraction of the retinal arteries and thickening of their perivascular lymph-sheaths, spots of degeneration in the retina, especially in the macula, represent the fifth stage, which passes into the final stage of so-called *papillitic atrophy.*

As the last stage is ushered in the borders of the disk begin to be visible, usually first upon the temporal side, until finally all margins again are apparent, at first a little mellowed, while the center is still covered by the remnant of the inflammatory tissue. Finally, the edges of the disk are clear, its color is white and atrophic, and its center becomes apparent. Both sets of vessels are contracted, and may be streaked along their sides with whitish tissue. Areas of retinochoroiditis and elevated patches of degeneration, marking spots of former hemorrhages, are often apparent. Second attacks of neuritis and choking of the disk may occur, as in a case observed by the author and A. G. Thomson. A choked disk may be implanted on an atrophic nerve-head.

In addition to the swelling of the disk, there may be marked *edema of the retina* and lines of edema in the macular region, forming the so-called *macular figure*, or *macular fan* (Paton), not unlike the appearance which is so striking in certain types of renal retinitis (see page 433). It occurs in a fair percentage of cases of cerebral and cerebellar tumors (fully 15 per cent. according to Paton's figures), and may reach a height equal to or greater than that of the choked disk. In addition to these areas of retinal edema, there may develop in the macular region yellowish-white and degeneration spots, intermixed with hemorrhages.

The *prognosis* of optic neuritis and choked disk depends upon the cause and the duration of the process. If, for example, syphilis is the active agent, there is reason to hope that suitable treatment will be followed by good results. If the focus of disease, for instance, in the accessory sinuses can be removed, vision may be saved and edema and inflammation will subside. If the papilledema depends upon increased intracranial tension, and this is relieved by decompressive trephining, or by a radical operation with removal of the growth, and the disk changes have not passed beyond the third stage, the prognosis as to sight is favorable. Untreated choked disk, or optic neuritis, almost always produces blindness; very exceptionally the original disease continues, but the neuritis subsides (Oppenheim).

Causes.—The most frequent cause of choked disk is *tumor of the brain*, inasmuch as it occurs in fully 80 per cent. of the cases. Usually the intracranial neoplasm must have existed for some time and the increased intracranial tension have lasted for a definite period before the engorgement-edema develops. The period from the beginning of choked disk to the height of its swelling in some instances comprises only a few weeks; in others, months and even years may elapse before the disk-edema appears. It is not possible to determine with certainty from the stage of the disk or retinal phenomena what the duration of the cerebral lesion is, but if choked disks arise with suddenness and the edema rapidly increases, they indicate an increase in intracranial pressure, either because the growth itself has gained in volume or because hemorrhage has occurred in or around it.

Tumors of the corpora quadrigemina give the highest percentage of choked disk, and next tumors of the parieto-occipital region and of the cerebellum, which yield an almost identical percentage (tables of John E. Weeks and J. M. Martin). Tumors of the basal ganglia are usually associated with papilledema. Choked disk, if it does not fail entirely as a symptom of tumor of the pons, of the medulla, and of the corpus callosum, is apt to be late in its development, and, to a certain extent, this lateness of development applies to tumors of the frontal and parietal convolutions. It is probable that pontine tumors give rise to choked disk only if they also involve some neighboring structure, and, according to Paton's researches, the bulk of cases of brain tumor without choked disk are those of pontine and subcortical origin, but if the subcortical growths spread to the base, the disk changes appear. Tumors of the cerebellum are prone to cause a more intense form of choked disk, with rapid depreciation of vision, than cerebral neoplasms, and the same intensity of the process is, according to some authors, evident in morbid growths of the midbrain and thalamus, while it is less pronounced in subcortical, parietal, and frontal lobe tumors. Whether the refraction of the eye has any influence on the development of choked disk is undecided. That myopia seems to have a deterrent effect has been asserted (Marcus Gunn, the author); its influence in this respect is doubted by other observers (Paton, Parsons, Bordley, and Cushing). The development of choked disk does not necessarily depend upon the size, situation, or structure of the intracranial neoplasm, and all types of morbid growths may originate papilledema—fibroma, sarcoma, glioma, carcinoma, solitary tubercle, and gumma.

It also appears with echinococcus cysts, epidural and subdural clots, intracranial trauma, abscess of the brain, and middle-ear disease, when this has extended to the cerebrum. Von Hippel describes a form of optic neuritis with affections of the ear which may exist without disturbing sight and remain even after operation has removed its apparent cause. *Acute blindness* with normal fundus due to the pressure of internal hydrocephalus caused by brain abscess has been described (Pagenstecher).

Of the four varieties of meningitis—simple, tuberculous, traumatic,

and cerebrospinal—tuberculous disease of the brain is the most frequent cause of optic neuritis, the percentage varying from 76 to 81 per cent. The appearances of the disk most often are those which have been described in connection with *descending neuritis* (see page 471). When there is direct pressure upon the tracts and chiasm, the swollen papilla has a peculiar grayish-white color, without much vascularity, and a similar appearance is sometimes caused by tumors of the cerebellum.

Other intracranial causes are softening of the brain, hemorrhagic pachymeningitis, cerebritis, hemorrhage, thrombosis of the cavernous sinus, hydrocephalus, aneurysm, and enlarged pituitary body in acromegaly.

Occasionally general paresis, epilepsy, and disseminated sclerosis are accompanied by optic neuritis and by choked disk. Papillitis may precede, accompany, or follow myelitis (ophthalmoneuromyelitis).

In addition to the intracranial causes of papillitis, this phenomenon may arise from a general infection. To this form Uhthoff gives the name *infectious optic neuritis*. According to this observer, it should be differentiated from those cases which are caused by orbital, intra-ocular, or intracranial lesions, and may be originated by any of the following diseases placed in order of their frequency: Influenza, syphilis, rheumatism, malaria, typhus fever, measles, whooping-cough, diphtheria, polyneuritis, small-pox, beriberi, erysipelas, scarlet fever, tuberculosis, typhoid fever, gonorrhea, and relapsing fever. The neuritis may manifest itself as a papillitis or as a retrobulbar neuritis, and Uhthoff thinks that the optic nerve conditions are most apt to arise during the stage of convalescence and are probably due to the action of toxins, and not directly to the micro-organisms. In children with congenital syphilis, even when only a few weeks old, optic neuritis is not uncommon, according to Mohr. Optic neuritis may also be caused by toxic agents, for example, by lead, atoxyl, filix mas, and alcohol, by anemia, both when this is an essential process and when it is caused by excessive hemorrhage, by disturbances of menstruation, by lactation, by exposure to cold, by myxedema, by sunstroke, and by injuries. An association of chlorosis, choked disk, and abducens palsy has been observed, attributed by Meller to thrombosis in the region of the cavernous sinus.

Metastatic optic neuritis has been recorded as occurring in sepsis (Axenfeld, von Michel). Optic neuritis may be associated with diseases and injuries of the anterior part of the eye. Under these conditions vitreous opacities may also be present.

Optic neuritis followed by atrophy may arise in association with *deformities of the skull*, and, according to Friedenwald's analysis, the patients for the most part have had oxycephalic or steeple-shaped skulls ("tower skulls"). Blindness without changes in the intra-ocular end of the optic nerve due to cranial deformity has been reported by C. A. Oliver. Papillitis occasionally occurs as a congenital affection in several members of the same family and sometimes appears without evident cause.

Choked disk and optic neuritis may depend upon disease of the

orbital region—inflammation of its contained tissues, tumors, caries, and periostitis, especially around the optic foramen, upon purulent disease of the antrum of Highmore and the frontal sinus, and morbid processes of the upper posterior portion of the nose and of the sphenoid and ethmoid bone (*sinusitis*). In most of these instances, unless both orbits or the sinuses are affected, the papillitis is unilateral, and there are other symptoms around the eye which point to the local condition. Optic neuritis has also been ascribed to dental disease.

A rare form of optic neuritis is that described in association with persistent dropping of a watery fluid from the nose. Headache, vomiting, unconsciousness, and delirium are present. The fluid has been believed to be identical with the cerebrospinal fluid (Leber) or to be

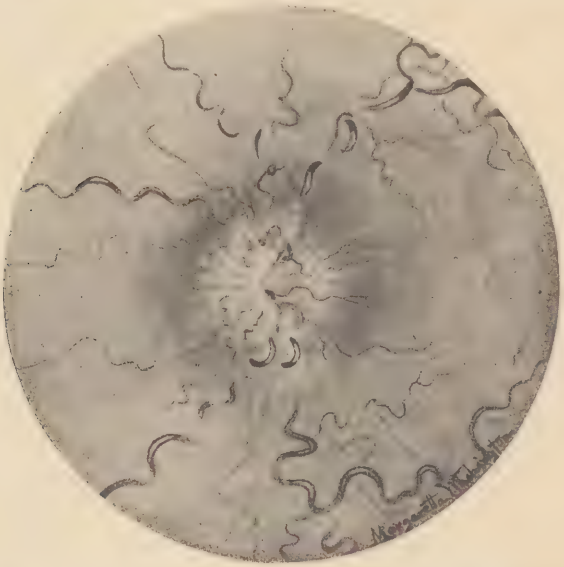


Fig. 215.—Fundus of the right eye of a patient with tumor of the brain and choked disk; swelling 6 D. (Service of Dr. Edward Martin in University Hospital.)

due to nasal disease in the form of small polypi (Nettleship and Priestley Smith). Internal hydrocephalus was present in some of the patients.

Treatment.—This depends upon the cause of the condition. In all syphilitic cases rapid mercurialization should be tried, followed later by the iodids. Salvarsan and neosalvarsan have been effectively employed. Orbital and sinus diseases indicate appropriate surgical measures.

Since Sir Victor Horsley's announcement, twenty-seven years ago, that the release of intracranial tension arrests and cures optic neuritis (choked disk), numerous operations have been performed, with satisfactory results. In a certain number of cases of brain tumor the growth can be removed by a radical operation, but even if it is inoperable, as it frequently is, or cannot be localized, a palliative operation,

that is, *cerebral decompression*, should be performed in order to save sight, and the earlier it is done, the better the results will be—*i. e.*, operation should be undertaken before the third stage of the disk change is reached. Usually, after technically correct operations (and in all pretentorial tumors temporal decompression is the operation of choice, and in all subtentorial lesions a suboccipital decompression is indicated [C. H. Frazier]), the choked disk begins to subside from the third to the tenth day, and the subsidence is complete at the end of six weeks. The same operation is urged by Cushing in choked disk caused by cerebral edema, infections, and intracranial hemorrhage. Choked disk has also been treated by lumbar puncture, but with this procedure for this purpose the author has had no experience, and it should be remembered that

Fig. 216.—Fundus of the right eye of the same patient shown in Fig. 215 one month after cerebral decompression.

lumbar puncture is not free from danger in the presence of intracranial tumor. *Puncture of the corpus callosum* and drainage of the ventricle have been utilized to reduce intracranial pressure and thus relieve papilledema.

Significance of Choked Disk.—Double choked disk is highly significant of intracranial disease, especially tumor or basilar meningitis. Indeed, it is the most important general symptom of this condition, but it is not a pathognomonic sign. The other causes of optic neuritis and choked disk which have been mentioned must be excluded, and care must be taken not to mistake the macular figure (see page 475) for an albuminuric retinitis. Although the presence of choked disk is so highly significant of cerebral tumor, of itself it possesses no distinct localizing importance. Usually papilledema is bilateral, but in a certain number

of instances it is unilateral, and frequently there is an excess of choking in one eye as compared with the other. Whether this is a sure indication that the tumor is on the same side as the choked disk or the excess of edema is undecided. Horsley believes that choked disk tends to develop earlier and is not more marked in the eye corresponding to the side on which the tumor grows, but Paton doubts if reliance can be placed on this sign. In the author's and Holloway's investigations in the majority of cases the greater swelling was on the same side as the tumor; but there were many cases in which this rule did not hold good. The development of choked disk does not depend upon the size nor on the type of the tumor, although according to some authorities disk changes are most frequently absent in tuberculous growths and most frequently present with sarcoma, glioma, and cysts. According to Walter R. Parker, choked disk caused by increased intracranial tension appears first in the eye with the lesser intra-ocular tension, as measured with the Schiötz tonometer. As this author himself points out, this observation requires confirmation.

Pathogenesis of Papillitis and Choked Disk.—As is well known, von Graefe at one time sharply distinguished between *descending neuritis* and *choked disk* (*Stauungs-papille*).

If, for example, in meningitis the sheaths of the optic nerve, which are continued over it as prolongations of the corresponding brain-membranes, participate in the inflammation, as they undoubtedly may, there is at first, as Greeff points out, a *perineuritis*, which extends by way of the connective-tissue septa to the trunk of the nerve. The evidences of this inflammation, soon visible to the ophthalmoscope, present the appearances of a moderate, that is, not engorged, intra-ocular optic neuritis, and the whole process is a *descending neuritis*.

If, on the other hand, the state of the nerve-head indicates engorgement, edema, and mechanical obstruction, and the evidences of these conditions are visible to the ophthalmoscope in the appearances already described (see page 474), the process is a *choked disk* or *papilledema*.

Inasmuch as ophthalmoscopically it is frequently difficult to distinguish a neuritis from a beginning choked disk, and as the conditions may be mixed, Hughlings Jackson expressed the opinion that there is one kind of optic neuritis from intracranial disease which may manifest itself under different appearances, sometimes with and sometimes without "swelling of the disk." It would seem, however, that it is still proper to maintain, within the limits described, the distinction to which reference has been made.

Numerous theories have been propounded to explain the *pathogenesis of choked disk*. Von Graefe believed that choked disk was due to a venous stasis occasioned by obstruction to the return of venous blood from the cavernous sinus. This theory ceased to be tenable when Sesemann demonstrated the anastomosis between the ophthalmic and the anterior facial veins. Parinaud taught that choked disk is due to extension of the interstitial edema of the brain tissue through the optic nerve to its intra-ocular end, a theory to which Sourdille subscribes; and

Kampherstein believes that often it can be explained only by a preceding edema of the brain, extending through the optic nerve to the lamina cribrosa and thus causing choking of the nerve-head.

The *inflammatory* or *toxin theory*, with various modifications, assumes, as Leber suggested and Deutschmann afterward endeavored experimentally to show, that so-called papillitis is not a product of edema, but an inflammatory affection, the fluid which distends the sheath of the nerve possessing an irritative quality; or, in other words, that the subarachnoid fluid is infected by products from the intracranial disease or lesion which is the prime cause of the trouble.

The *mechanical* (*lymph-space*) *theory* of Schmidt-Rimpler, ascribed to the dropsy of the intersheath space of the optic nerve, which is caused by the increased subarachnoid fluid being forced into this situation under the influence of elevated intracranial pressure, a mechanical or compressing action, or to the fluid which found its way into the lymphatic spaces of the optic nerve, an action causing edema, congestion, and later inflammation.

Merz, trusting to experimental evidence, which has not always been confirmed (Frazier, J. E. Sweet, Holloway, and the author), declares that increased intracranial tension alone is sufficient to produce choked disk and that it is only necessary that this tension shall be maintained uninterruptedly for a certain time. Cushing and Bordley have, repeating and elaborating Merz's experiments, reached similar results, and believe that a mechanical rather than a toxic process plays the chief rôle in the causation of choked disk. That the disk changes are due to compression of the vessels as they pass through the lamina was originally maintained by Manz. Choked disk, according to Schieck, is due to lymph stasis, the cerebrospinal fluid passing by way of the perivascular lymph-sheaths of the axial bundle of the optic nerve and along the central vessels into the disk. Paton and Gordon Holmes believe that their observations establish the fact that papilledema is an edema of the nerve-head due to two factors—venous engorgement and lymph stasis.

In general terms it is probable that choked disk is produced by a combination of factors. In this combination increased intracranial tension or pressure is most prominent, and the mechanical theory of its pathogenesis affords the most satisfactory explanation. If other factors are potent, they have not yet been definitely discovered.

Certainly, inflammatory or irritative processes in the optic nerve and its sheaths sometimes have an active influence, and if the inflammatory condition predominates, the elevation of the disk is less marked and the process is apt to extend to the retina, where exudations and hemorrhages are visible; in other words, the lesions warrant the descriptive term *inflammatory optic neuritis*.

Pathology.—Sections of choked disk examined with the microscope reveal the following lesions: Edema and swelling of the nerve-head, blood extravasations, swelling and varicosities of the nerve-fibers, and slight cellular exudation along the thickened and dilated vessels. In the interstitial form of neuritis the inflammation begins in

the sheath and septa, with the formation, in addition to the edema, of an exudation rich in cells, which subsequently organizes. There follow thickening of the interfascicular septa, increase of the nuclei, and degeneration and atrophy of the nerve-fibers from pressure. In some cases degeneration of the ganglion cells of the retina is evident, depending upon the fact that an arterial branch supplying that particular area has been occluded. Such degenerative areas may give rise to scotomas or sector-like defects in the visual field. An ampulliform dilatation of the optic nerve sheath posterior to the eyeball is found in a certain number of cases, and in addition to distention of the intervaginal space there may be an infiltration of small cells in the sheath.

1. **Optic-nerve Atrophy.**—Under the general term *atrophy of the optic nerve* are included the various types of degeneration and shrinking of the fibers of the optic nerve, usually described under the subdivisions *primary*, *secondary*, *consecutive* (*neuritic* or *postpapillitic*), and *retinal* and *choroiditic atrophy*. The last are really forms of consecutive atrophy.

Symptoms.—Certain general symptoms are common to optic-nerve atrophy, although these are subject to variations according to the clinical types.

1. *Changes in the Nerve-head.*—(*a*) *Alterations of the Normal Color of the Disk.*—The color of the disk varies from a slight gray pallor to a pure gray, greenish-gray, or entirely white hue. Many intermediate forms of discoloration occur; thus there may be a commingling of gray and red, producing the so-called "gray-red disk," and often there is a decided greenish tinge, rarely a blue one.

Grayness of the optic nerve is not always detected by ordinary methods of examination, especially in the deeper layers of the disk, but if the fundus is examined by means of properly regulated illumination, and through a lens which neutralizes any error of refraction, this deep pallor is usually evident. It is important to employ both the direct and indirect methods of examination, and the concave and plane ophthalmoscopic mirror.

(*b*) *Alteration in the Center of the Disk.*—Sinking of the surface of the disk, varying from a slight depression to a complete excavation (see page 370), occurs according to the degree of degeneration which the nerve-fibers have experienced. The shape of the excavation depends somewhat upon the presence or absence of a normal physiologic cup. At the bottom of the atrophic excavation the mottling of the lamina cribrosa is very distinct in some cases of atrophy; in others it is not apparent.

(*c*) *Alterations of the Margins of the Disk and of the Scleral Ring.*—In complete atrophy the margin of the optic disk is unusually distinct. In the atrophy which follows a neuritis or retinitis, however, the margins are often slightly veiled for a long time.

Undue broadening of the scleral ring indicates shrinking of the disk. Even in the early stages of spinal atrophies the disk may be surrounded by a broad scleral ring, which, taken into consideration with alteration in the color of the papilla and contraction of the color-field (especially

red and green), affords diagnostic aid in the study of gray degeneration of the optic nerve.

2. *Changes in the Vessels.*—In simple atrophy, while there may be narrowing of the vessels, this is not always the case, and certainly not in the manner seen in consecutive atrophies. Sometimes the arteries are narrowed and the veins unchanged.

In neuritic (consecutive) atrophy the arteries are much contracted and the veins in contrast are larger than usual, often retaining some of the tortuosity which was so marked a feature during the papillitic stage. By the contraction of the tissue these, too, may later become narrowed. Development of white tissue along the course of the vessels, due to thickening of the perivascular lymph-sheath, is common in this form of atrophy.

In retinitic and choroiditic atrophy there is marked contraction of both veins and arteries, which at the same time are diminished in number.

3. *Changes in the Surrounding Eye-ground.*—The presence of alterations in the general fundus depends entirely upon the cause of the atrophy. In simple gray and white atrophy such signs are absent; but in postpapillitic and retinitic atrophy, spots of degeneration, marking the places of former hemorrhages, and patches of pigment heaping, are commonly seen.

In addition to these ophthalmoscopic changes the following symptoms occur:

1. *Change in Central Vision.*—This varies from a slight depreciation to blindness, and, if the atrophy is bilateral, is usually more marked in one eye than in the other. In every case, where this is possible, especially in early cases or cases of doubtful atrophy, any existing refractive error should be corrected before deciding the degree of depreciation of central sight.

2. *Change in Light-sense.*—Usually it has been found, in pure optic-nerve atrophy, that the light-difference is increased, but the light-minimum not much influenced. P. F. Hay, however, has also observed considerable increase in the light-minimum. *Adaptation,* or the accommodation of the eye to varying degrees of illumination, is greatly restricted, for example, in tabetic optic-nerve atrophy (Lohmann).

3. *Change in the Field of Vision for Form* (*White*).—The following changes occur: Concentric contraction; very irregular limitations presenting large re-entering angles (peripheral scotomas); quadrant-shaped defects; complete loss of one-half of the visual field (hemianopsia); and an abnormal blind-spot in the center of the field (central scotoma) or adjacent to it (paracentral scotoma).

The field of vision, concentric restriction being most common, does not give evidence of the cause of the atrophy, although it may afford information of the localization of the defect; thus, an affection of the macular fibers will produce a central scotoma. In spinal atrophy the limitation more frequently begins at the outer side than in other situations.

4. *Change in the Field of Vision for Colors.*—There is always a defect in color vision. Usually there is, first, contraction of the green field, then of the red, and afterward of the blue and the yellow fields. In late stages of optic-nerve atrophy color-sense is abolished. Occasionally the perception of red becomes defective before that of green is influenced by the atrophic process.

Generally the contraction of the color-field is much greater than that of the form-field (white) (compare page 386). Central vision may be good, the form-field but slightly or not at all affected, and yet the green and the red fields may be considerably contracted. Hence the importance of combining all these examinations before deciding whether discoloration of the papilla is pathologic or not.

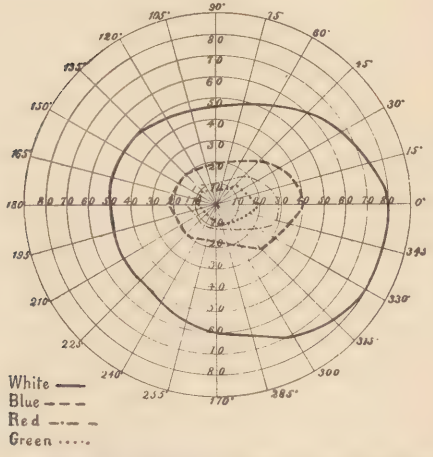


Fig. 217.—Field of vision of the right eye in a case of optic-nerve atrophy. The form-field is slightly contracted, the color-fields markedly restricted (compare Fig. 43, page 84).

4. *Changes in the Pupil.*—The relations of the pupil to the action of light depend upon the degree of atrophy. In many cases there is more or less perfect paralytic mydriasis, and when the atrophy is complete the pupil is dilated and the iris motionless. Even when the pupil fails to contract under the influence of light thrown upon the retina, it may do so in the act of convergence. (See also page 63.)

If the atrophy is confined to one eye, no reaction will occur when the light falls upon the corresponding retina, but instant contraction takes place when this is directed upon the retina of the opposite (unaffected) side. The pupil changes in spinal disease (tabetic atrophy) have been described (see page 63).

Varieties of Optic-nerve Atrophy.—1. *Primary Atrophy (Sometimes called Gray, Progressive, Spinal, or Tabetic Atrophy).*—The color of the disk is gray or white; sometimes it has a greenish or bluish tint; the discoloration is associated with translucency, and the stippling of the lamina is evident; the excavation, if it exists, is complete and saucer-like; the vessels either are smaller than normal, especially the arteries, or they are unaffected in size; the edge of the disk is sharply marked, and the scleral ring clean cut all around. These symptoms describe the fully formed atrophy.

In the earlier stages of the degeneration, according to the late Dr. W. F. Norris, the disks are of a dull red tint, their capillarity is superficial, and the deeper layers, in the neighborhood of the lamina cribrosa, are gray and wanting in circulation. There is often sufficient haze of

the retinal fibers to veil the scleral ring. Later the nerves become pallid, are somewhat woolly superficially, and are surrounded on all sides by broad and sharply cut scleral rings. The larger retinal arteries and veins do not at this stage present any appreciable change in their caliber or appearance. Both eyes usually are affected, one showing a further advance of the degenerative process than its fellow.

2. *Secondary Atrophy.*—The color of the disk may be gray and assumes a tint not greatly dissimilar from the atrophy which has just been described. In other instances the color is more decidedly white. Both sets of vessels may be contracted, usually the veins being less affected than the arteries. In a certain number of cases of secondary atrophy it is probable that preceding the degenerative stage there is a transient congestion of the disks; certainly this is true in those cases where there has been a retrobulbar neuritis.

3. *Consecutive Atrophy.*—(*a*) *Postpapillitic Atrophy.*—The color of the disk is very gray or white, sometimes with a decidedly greenish tinge or even a blue tint. It is noticeable, however, that the translucency present in the primary form of atrophy is absent, and the stippling of the lamina cribrosa is not visible, owing to the existence of a non-transparent tissue which covers it. The borders of the disk are slightly veiled, and the perivascular lymph-sheaths are thickened. The arteries are contracted, the veins frequently exhibiting distinct tortuosity. Retino-choroidal changes are often evident.

(*b*) *Retinitic and Choroiditic Atrophy.*—This is in the form of atrophy of the nerve to which reference has already been made, and which follows severe forms of retinitis and choroiditis. The disk has a distinctly yellowish tinge, being somewhat waxy in appearance; its borders are not sharply marked, and the vessels are narrowed, often to a great degree.

Causes.—In addition to the forms of atrophy which follow inflammation of the nerve (*postpapillitic atrophy*), inflammation of the choroid and retina (*choroiditic* and *retinitic atrophy*), embolism and thrombosis of the central artery and thrombosis of the central vein of the retina (*embolic* and *thrombotic atrophy*), the varieties which are gathered under the general terms *primary* and *secondary* atrophy require mention.

Gray degeneration of the optic nerve occurs, in the great majority of instances, under the influence of diseases of the spinal cord, and especially of locomotor ataxia. It is frequent in general paralysis of the insane and insular sclerosis, but less common in lateral sclerosis. There is some difference of opinion in regard to the frequency of optic-nerve atrophy in locomotor ataxia, but an average of a number of observations gives 33.7 per cent. of atrophies. In most instances it begins in the preataxic stage. To one variety of the affection, in which the atrophic process precedes, often by long intervals, the ataxic symptoms, the name *optic-nerve type of tabes dorsalis* is often applied. Optic-nerve atrophy has also been seen with Friedreich's ataxia, amyotrophic lateral sclerosis, chronic myelitis, paralysis agitans, spastic spinal palsy, and bulbar palsy.

Primary atrophy has been ascribed to the influence of cold, imperfect nutrition, disturbed menstruation, and venereal excesses. It may be caused by diabetes, syphilis, the toxic action of certain drugs, and excessive hemorrhage (see also page 501). Its association with deformities of the skull has been described (see page 477). Undoubtedly it may be due to arteriosclerosis, the thickening of the arterial wall closing the lumen of the vessels.

Hereditary Optic-nerve Atrophy.—A remarkable type of optic-nerve atrophy, that is, of the papillomacular bundle, first systematically described by Leber (*Leber's disease*), is hereditary, and may appear for a number of generations usually, but not always, in the male members of the family. It is transmitted by unaffected females. An affected male seldom transmits the disease (Nettleship). The disease generally begins between the eighteenth and twenty-third year, but has been observed as early as the fifth year and delayed as late as the forty-third year. According to Norris, there are three stages of the affection: (1) Stage of edema and congestion of the disk; (2) stage of gray discoloration of the nerve-head; and (3) stage of pronounced atrophy. Central scotomas are commonly present. In Arnold Knapp's cases the first generation presented central scotomas and peripheric contractions of the visual fields; but in the second and third generations no central scotomas developed. Gould suggests "homeochronous hereditary optic-nerve atrophy" as a suitable name for the disease. Usually treatment has little or no effect, but improvement and even recovery have been reported (Cargill).

Secondary atrophy appears under the influence of compression of the optic tract and the optic fibers—for instance, by bulging of the lateral ventricles, or by pressure of a tumor, pituitary neoplasm or struma, exostosis, or aneurysm upon the chiasm. It is also said to occur with meningitis without preceding neuritis. Compression around the optic foramen is likely to produce secondary atrophy by direct injury to the fibers of the optic nerve. Blows on the head, especially in the neighborhood of the supra-orbital foramen, causing fracture of the orbital plate or periostitis, may be followed by a like result. Atrophy may result from inflammation of the axis of the nerve posterior to the eyeball.

Pathology.—In simple degeneration as it occurs in tabes of the cord the nerve-fibers lose their medullary sheaths and are converted into fine fibrillæ, between which are numerous fatty granular cells; no true inflammatory process appears. Later all nervous elements may disappear. Tabetic atrophy of the optic nerve has been ascribed to disease and disappearance of the retinal ganglion cells (Ward Holden), but from a comparison of the visual fields in glaucoma with those in tabetic atrophy Rönne concludes that the lesion is in the optic nerve-fibers, and Stargardt seems to have demonstrated that an exudative process in the chiasm and nerves precedes the degenerative one. In postneuritic atrophy there is considerable new-formed connective tissue in the nerve-head and trunk, through which run the thickened vessels; the sheaths of the nerve-fibers degenerate, break down into fine drops,

PLATE VII.

Primary atrophy of the optic nerve.

and the nerve-fibers become varicose and either shrink or disappear. The septa are much thickened, and in advanced cases the nerve becomes a narrow, purely connective-tissue cord.

Diagnosis.—The student is warned not to mistake the pallor of age for the pallor of disease; not to mistake a large physiologic cup, with its margin shelving toward the temporal border of the disk, for an atrophy confined to half of the optic papilla; and not to mistake small patches of retained marrow-sheath for atrophic changes.

Not every gray disk, with an unusually marked scleral ring, is indicative of atrophy, and it is only when these appearances accord with the other manifestations of beginning degeneration that the diagnosis of incipient atrophy is justified. The Wassermann test should always be employed, and a stereoscopic *x*-ray plate should be made in order to determine the condition of the pituitary region and the accessory sinuses of the skull.

The differential points between a chronic glaucoma and an optic-nerve atrophy have been described (see page 386), and also the relation of light-sense to optic-nerve atrophy. According to Lohmann's investigations all those cases with a disproportionately great affection of *adaptation* appear to belong to the group of glaucomatous degenerations in contrast to other forms of atrophy.

Course and Prognosis.—The course of optic-nerve atrophy is always a slow one, lasting for months and it may be years, depending to a certain extent upon the original cause of the atrophy.

The prognosis is unfavorable in primary or, as it is sometimes called, progressive atrophy, the tendency being to a gradual deterioration of sight with shrinkage of the field of vision, until complete blindness is the result. The prognosis of a consecutive atrophy depends entirely upon the amount of damage which is likely to ensue from the shrinking which follows during the subsidence of the neuritis. In the forms of atrophy which follow an inflammation of the axis of the nerve the prognosis is better.

Treatment.—This depends upon the cause. If syphilis is present, the usual remedies are indicated; but mercury is useless in advanced cases, even in syphilitics. While salvarsan or neosalvarsan exercise no detrimental effect on a healthy optic nerve, at one time it was maintained that salvarsan exerted an evil influence on tabetic atrophy. Recently this contention has been largely set aside and there are now a number of observations on record which tend to show that neosalvarsan, if used early and while color perception is still good, represents a therapeutic agent of value in the treatment of tabetic atrophy. *Salvarsanized serum* injected intraspinously (Swift-Ellis method) may do good if employed before the degenerative process has begun. Strychnin has been much employed administered in full doses, preferably by the hypodermic method; it may be enforced by nitroglycerin. Other remedies, according to the cause, are iodid of potassium, nitrate of silver, phosphorus, arsenic, iron, santonin, lactate of zinc, hypodermics of antipyrin (Valude), and injections of organic liquids, all of doubtful value. Negative gal-

vanism has been advised, and good results have been reported by L. W. Fox, Ziegler, Radcliffe, F. W. Coleman, and many other observers. Coleman especially recommends the *sinusoidal current*, a binocular electrode being placed over the eyes and an oval pad to the nape of the neck. The treatment should take place for twenty minutes each day. High-frequency currents have been advocated and they should be tried. In a few instances suspension is said to have been followed by improvement of vision in tabetic atrophy. There is no satisfactory evidence that radium and the Röntgen rays are useful therapeutic agents in the treatment of optic-nerve atrophy.

Orbital Optic Neuritis (*Retrobulbar or Axial Neuritis; Central Amblyopia*).—In contradistinction to the optic neuritis which is specially localized at the intra-ocular end of the nerve, an inflammation occurs in the orbital part of the optic nerve, which is called *orbital optic neuritis—retrobulbar* or *axial neuritis*. It appears in an *acute* and a *chronic* type.

1. *Acute Retrobulbar or Axial Neuritis.*—The symptoms of this affection are the following: Obscuration of vision, beginning always in the center of the visual field, and rapidly progressing in from one to eight days to complete or nearly complete blindness; at first negative ophthalmoscopic appearances, later blurring of the margins of the disk, hyperemia of its surface, and sometimes, in severe cases, diminished caliber of the retinal arteries and fulness and pulsation of the retinal veins; distinct pain on movement of the eyeball, or when the globe is pressed backward into the orbit.

The affection appears to depend upon an interstitial neuritis, most severe in the optical canal, and at first chiefly located in the papillomacular tract, from which it may extend, however, until the whole diameter of the nerve is involved. If the process is unchecked, necessarily secondary degeneration of the nerve-fibers takes place. There is also degeneration in the ganglion cells of the macula.

Cause.—The determining cause of the disease is the presence in the blood of an infecting agent existing in association with some disease—for example, rheumatism, tuberculosis, gout, syphilis, influenza, diabetes, small-pox, and scarlet fever; or coming directly from a focus of infection in the mucous membrane of the nose, the mouth (the teeth), the tonsils, the ethmoid cells, and the sphenoid sinus; or arising as the direct result of an inflammatory process in the orbit—*e. g.*, cellulitis, or in the optic canal—for example, periostitis, gummatous deposits, etc. The disease has also been attributed to certain toxic agents, such as alcohol, lead, etc.; to menstrual disturbances, especially sudden suppression of the menses, to auto-intoxication, and to overwork and prolonged eye-strain. A certain number of cases exist for which no cause can be ascertained. Nettleship divided cases of retrobulbar neuritis into two groups: the *idiopathic*, in which the disease starts in the nerve itself, and *symptomatic*, in which it is communicated to the nerve by the surrounding tissues.

This disease may be part of the symptomatology of multiple scle-

rosis and of acute or subacute myelitis, and is in these circumstances of most serious prognostic import. Retrobulbar neuritis ipsolateral to the lesion has been recorded as a symptom of tumor or abscess of the frontal lobe (F. Kennedy). The author has observed several cases of this character.

The *course* of the disease may be rapid or fulminant, as it is called. It is sometimes bilateral, but more frequently unilateral, or a long interval may occur between the involvement of the first and second eye. Relapses may occur, and the affection may alternate between the two eyes. As pointed out by Mr. Marcus Gunn, there is marked analogy between axial inflammation of the optic canal and paralysis of the facial nerve (Bell's palsy) when its trunk is involved in its tortuous course through the wall of the skull. Indeed, as the author has shown, retrobulbar inflammation may be preceded by an attack of peripheral facial palsy, either upon the same or the opposite side.

Although the *prognosis* must always be guarded, in the majority of instances the tendency is to recovery, and, under careful treatment,

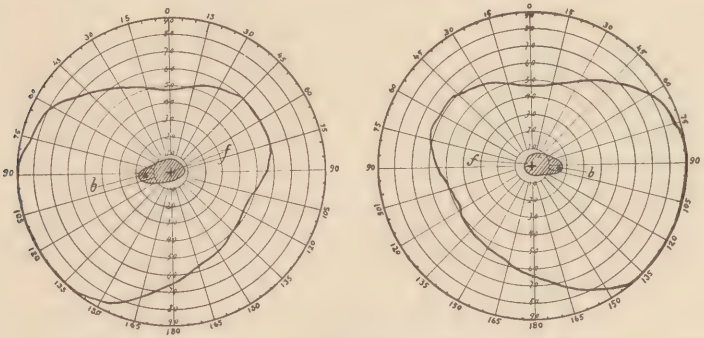


Fig. 218.—Central scotoma from a case of tobacco amblyopia: *f*, Fixation; *b*, blind-spot.

to perfect recovery. In severe cases, permanent pallor of all or part of the optic nerve, defective central vision for colors, central scotoma, and contraction of the peripheral field may remain. The fact that retrobulbar neuritis may indicate the future onset of disseminated sclerosis should not be forgotten. It may precede the other symptoms by many years. According to Marx, it may develop in a certain percentage of the cases from one to seven years after apparent recovery. There is also a variety of the disease due to exposure, menstrual disturbances, and rheumatism, in which the same symptoms appear as those previously described, but all of a milder type and all more amenable to treatment.

Treatment.—In so far as possible the patient must be removed from the influence of the cause. If the affection has occurred during the course of an acute infectious disease, the treatment of this particular malady is indicated. In other circumstances the best results follow active diaphoresis, full doses of salicylic acid, the free use of mercury, the iodids, and counterirritation on the temple. No patient

with retrobulbar neuritis should escape searching examination of the nasopharynx, the ethmoid, frontal, and sphenoid sinuses. If purulent disease is found, operation with suitable drainage may speedily relieve the ocular condition; if this is neglected, blindness may result. (See also page 589.) Retrobulbar neuritis of tuberculous origin has been relieved by injections of tuberculin (Igersheimer).

2. *Chronic Retrobulbar or Axial Neuritis* (*Tobacco Amblyopia; Toxic Amblyopia*).—The clinical symptoms of this affection are as follows: Diminution of sight, associated with fogginess in the center of the field of vision, unimproved by glasses and most noticeable in bright light; reduced acuteness of vision, which varies from $\frac{6}{9}$ to counting fingers; negative ophthalmoscopic appearances or pallor of the temporal half or of a quadrant-shaped portion of the papilla; normal peripheral boundaries of the field of vision; symmetric central color scotomas, especially for red and green, usually oval in shape, stretching from the fixing-point to the blind-spot, and rarely passing much to the nasal side of the former; defective light-sense. The scotoma, which is the most important of the symptoms, represents a red-green blind area, and commonly the extent of green-blindness is greater than that of red, which, in its turn, may be surrounded by an area of imperfect color-sense. Sometimes its beginning is a small, easily overlooked scotoma exactly over the fixing-point (Groenouw). After the typical egg-shaped scotoma is developed, the process may cease, or there may be a stage of progression characterized by an increase in the size of the color defect, usually above, until it meets the limit of the red field; that is, the scotoma has "broken through." In severe cases there may be scotoma for blue and yellow. Finally, small absolute defects may be found, and in neglected cases, or in those not typically toxic, the entire scotoma may become absolute. The periphery of the visual field is not always intact, and contractions may be found if the tests are made under diminished illumination. According to Dorrell and Herbert Fisher the sensory pupil reflex is much diminished or entirely absent in toxic amblyopia.

Causes.—The most important drugs and toxic substances which may be responsible for the clinical symptoms which have just been detailed are tobacco and alcohol, either singly or combined, stramonium, cannabis indica, chloroform, chloral, opium, bisulphid of carbon, nitrobenzol, arsenic, atoxyl, lead, iodoform, and the toxin of diabetes. Of the substances mentioned, tobacco is the one most often responsible for this affection, but as the users of tobacco are also usually consumers of alcohol it is difficult to separate the etiologic influence of these two drugs, and hence the name *intoxication* or *toxic amblyopia* is used to describe a central amblyopia which may be due to either of these substances or to their combined influence. A pure tobacco amblyopia, which the author believes he has observed, is uncommon; indeed, some observers deny its existence. Although usually bilateral, a few instances have been recorded in which the symmetric development of tobacco amblyopia has been delayed. It is rare before the thirty-fifth year.

The pathologic lesion which causes this form of amblyopia, according to Uhthoff and other observers, is an interstitial inflammation of the *papillomacular* fibers of the optic nerve. These fibers, traced by means of their degeneration, consist of a bundle shaped like a triangle near the eye, with its base in the lower and outer part of the nerve, and its apex at the central vessels. Gradually it passes to the center of the nerve, which it reaches in the optic canal. Finally, it can be followed into the chiasm and tracts. Nuel and others believe that central toxic scotoma is not caused primarily by a neuritis of the macular bundle, but represents a disease of the macula lutea, causing degeneration of its cells, and that the optic nerve changes are secondary to destruction of the nerve-cells in the macula. The investigations of Birch-Hirschfeld lead him to doubt that the process depends upon a primary interstitial inflammation of the optic nerve. He believes that there is a primary involvement of the nervous elements of the nerve and retina, with an accompanying proliferation of the glia and increase in the

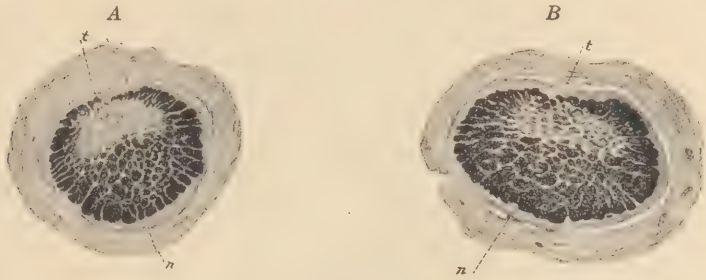


Fig. 219.—Sections of the right optic nerve in a case of toxic amblyopia, showing degeneration of the papillomacular bundle (Weigert's stain): *A*, Transverse section of the optic nerve 13 mm. behind the globe; *B*, transverse section of the optic nerve in the region of the optic foramen.

connective tissue. Schieck concludes that the disease begins with an alteration of the blood-vessels within the optic nerve, and, as the central fibers are less liberally supplied with blood, they are the first to be affected. While vessel disease may have an important bearing on this affection, a direct action of the toxin on the nervous elements seems undoubted.

Course and Prognosis.—The course is, as its name indicates, a chronic one, but the prognosis of the tobacco and alcoholic cases is good, provided the patients present themselves at an early enough stage for treatment. In rare instances complete optic-nerve atrophy results.

Treatment.—This consists in total abstinence from the use of tobacco and alcohol, and in the earlier stages this alone will be sufficient to bring about a cure. Later, a remedy of value is strychnin, which, as in other instances of optic-nerve disease, should be pushed to its full physiologic limit. In order to help in the absorption of inflammatory products, iodid of potassium may be given. Regulation of diet and free diaphoresis are valuable measures. Examination of the urine, as the author

and David Edsall have shown, is apt to reveal an excessive secretion of enterogenous decomposition products, and with its restoration to normal, under the influence of proper dietetic regimen, the eye conditions improve. The patients should drink water freely. Temporary improvement occurs under the influence of inhalations of nitrite of amyl, and the circulation of the optic nerve may be stimulated by the exhibition of digitalis and nux vomica. Lecithin has been recommended in the treatment of tobacco amblyopia (H. de Waele).

Necessarily, if some poison other than alcohol or tobacco is active, the patient must be removed from its influence.

Injury of the Optic Nerve.—This may be produced by the entrance of a foreign body into the orbit, for example, the end of a sharp stick, or from a fracture involving the bony wall of the orbit or base of the skull. Atrophy of the optic nerve is the result. Indirect injury of the optic nerve may be caused, as Evans has shown, by a blow in the region of the external angular process of the frontal bone. The primary impairment

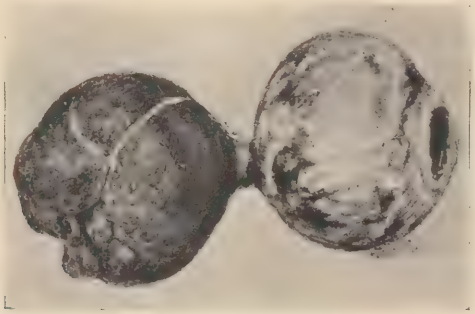


Fig. 220.—Intradural tumor of the optic nerve.

of vision and loss of the temporal field may be followed by atrophy of the nerve.

Tumors of the Optic Nerve.—These may be divided into *intradural* and *extradural* tumors. Of the former, 102 cases have been collected by W. G. M. Byers in his notable monograph on this subject, and they include fibroma, sarcoma, glioma, endothelioma, gumma, tubercle, and myxoma.[1] Of the latter (extradural tumors) Parsons has been able to find 12 undoubted cases, and of these, 9 were almost certainly endotheliomas.

The *symptoms* are: Exophthalmos, the eye being pushed downward and forward, the motion of the globe being unaffected, and defective vision, which is an early manifestation. The growth is slow and painless, but sometimes a suppurative keratitis may result. The ophthalmoscope reveals distended veins, edema, and choking of the papilla, followed later by white atrophy and shrinking of the vessels. Atrophy

[1] Many cases have been recorded since this publication appeared. The subject has also been elaborately investigated by Finlay, T. F. Bullar, and C. Devereaux Marshall. The author has observed psammo-endothelioma of the optic nerve and another tumor composed of fibrous tissue, neuroglia, and apparently ganglion cells.

of the papillomacular bundle and central scotoma may occur, as in one of the author's cases. With intradural tumors the movement of the eye is usually restricted in the opposite direction to any modification of the proptosis directly forward (Parsons).

Treatment.—In most instances the eyeball must be removed with the tumor, but occasionally the globe can be saved (11 times in Finlay's collection—subsequent loss in 4 cases). Exenteration of the orbit has been necessary, primarily, in a few instances, and secondarily on account of local recurrence. Even after enucleation, and sometimes at a period long removed from the time of operation, the growth has recurred and death may occur from intracranial involvement. Since the intro-

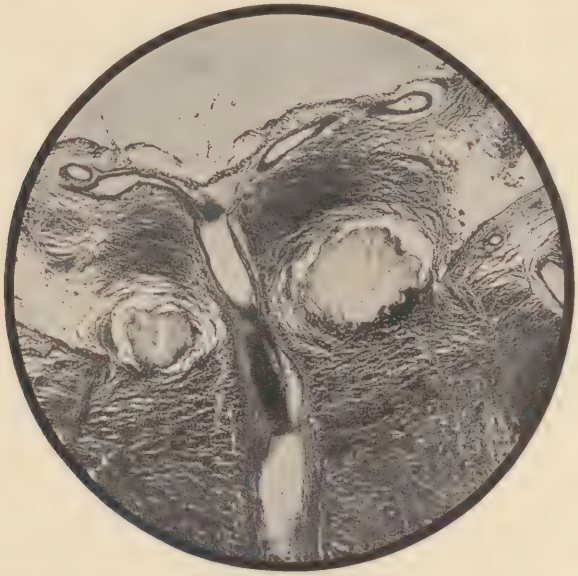


Fig. 221.—Microscopic section of a nerve-head containing hyaline bodies (from a photomicrograph).

duction of Krönlein's operation and its modifications the opportunity of exploration of the orbit has been greatly facilitated and the opportunity of saving the eyeball has been increased.

Hyaline Bodies (Drusen) in the Papilla.—This affection is characterized by the formation in the optic papilla of small excrescences or globular formations, which are sometimes described as colloid masses. The bodies are variously shaped, chiefly roundish, of a yellowish-white or bluish-gray color, forming a mulberry-like appearance and presenting a striking ophthalmoscopic picture. They may occur at any age of life, sometimes in association with choroidoretinitis, optic neuritis, and optic-nerve atrophy, but also in eyes free from other pathologic changes and with perfectly normal vision. Two views have been maintained in regard to the origin of the drusen: (1) That they are hyaline excrescences of the lamina vitrea of the choroid which become embedded in

the head of the optic nerve, and (2) that they have nothing in common with the choroidal excrescences, but are a special pathologic process confined to a small portion of the optic nerve. The microscopic studies of the author indicate that the latter view is the more nearly correct of the two. The exact nature of the material thus deposited has not been determined. One investigation by Hirschberg and Cirincione indicates that the bodies are amorphous and organic, and their composition appeared most to resemble that of elastin. They may undergo calcification, like the cheesy nodules in the lung. According to Parsons, "Drusenbildungen" upon the optic disk represent exudations which have been laid down in layers.

CHAPTER XVII

AMBLYOPIA, AMAUROSIS, AND DISTURBANCES OF VISION WITHOUT OPHTHALMOSCOPIC CHANGES

Amblyopia and amaurosis are terms which signify *dimness of vision*, the former being used to describe *obscurity of sight*, and the latter the more advanced condition of *loss of vision*. Although these terms usually describe defective vision unexplained by lesions in the eye, this limitation is not strictly followed, and eyes blinded by inflammatory disease are sometimes described as *amaurotic*.[1]

Modern methods of examination have greatly lessened the number of conditions to which the older writers applied the words "amblyopia" and "amaurosis." Amblyopia is a symptom and describes the defective vision from which the patient suffers. This may be due to functional disturbance or to disease of the visual apparatus (retina, optic nerve, or visual centers), and may be unassociated with changes in the eye-ground; or there may be atrophy of the optic nerve.

Amblyopia may be congenital or acquired; temporary or permanent; symmetric or non-symmetric.

Congenital Amblyopia.—This term is used to describe that variety of defective vision which for the most part is uncomplicated with fundus lesions, although sometimes the papilla is discolored and the macula deeply pigmented, and there is a scotoma, either small and absolute, or larger and for colors alone. According to Heine, a central scotoma can be demonstrated in 90 per cent. of the cases. The faulty vision has existed from birth, and often high grades of refractive error, especially hyperopia and astigmatism, are present, and clear images have never been focused upon the retina. Correction of the optical error usually fails to improve, materially, the vision; the retinal images continue to be defective. In very young patients an eye of this character may occasionally be trained to more nearly perfect vision after a proper correction of the refractive error, and this attempt should always be made.[2] Naturally, before the diagnosis of congenital amblyopia is established, the possible influence of orbital, nasal sinus, and central nervous disease must be eliminated. The origin of congenital amblyopia is obscure; an anatomic basis for the condition has not been discovered (Heine, Lohmann).

[1] The term "amaurosis" is also applied to certain cases of blindness in young children dependent upon hereditary influence, syphilis, tuberculous disease, and meningitis. The eye-grounds may or may not be diseased.

[2] A form of amblyopia has been described by Martin and called *astigmatic amblyopia*, dependent upon an imperfect development of the functions of the finer anatomic elements of the retina. It has been attributed to the fact that at the time of the education of the sense of sight, owing to astigmatism the retina has been asymmetrically stimulated, and consequently there has been asymmetry of visual acuteness.

Defective vision, attributed to lack of use (*amblyopia ex anopsia, argamblyopia,* according to Gould), may occur on account of obstruction to the rays of light falling upon the retina—*e. g.*, congenital corneal opacities, congenital cataract, and impervious persisting pupillary membrane; or in an eye which from early infancy has squinted, and has, therefore, not been concerned in the visual act (compare with page 542). The amblyopia of a squinting eye may disappear if the seeing eye becomes blind or is removed, as in a remarkable case reported by W. B. Johnson.

Gould maintains that certain cases of amblyopia which have been attributed to disuse are really due to a low grade of choroidoretinitis affecting the macular region, brought into existence by an irritating stimulus with which a long-continued ametropia has supplied this area.

In this category of amblyopias are also placed certain congenital defects of structure—*e. g.*, coloboma of the iris and deficient development of the entire eye (microphthalmos). Retinal hemorrhages in the newly born explain some cases. Usually one eye is affected; if both are amblyopic, nystagmus commonly is present.[1]

Congenital Amblyopia for Colors (*Color-blindness*).—Congenital disturbance of the color-sense has been found in about 3 per cent. of the examinations made for this purpose, but it is extremely rare in women (0.2 per cent.). Both eyes, except in rare instances, are affected, and a distinct hereditary tendency has been noted in many instances. In other respects the functions of eyes which are "color-blind" are normal, and the cause of the condition has not been determined.

The methods of detecting color-blindness have been described on page 67. Congenital color-blindness must not be confounded with the various disturbances of the color-sense in diseases of the optic nerve and retina or in hysteria.

Derangements of the perception of colors have been divided into two varieties: the one characterized by an absence of the power to perceive colors, or *achromatopsia;* and the other characterized by difficulty in distinguishing colors, or *dyschromatopsia.* The former condition, or color-blindness, is rarely *total* as a congenital defect (a condition which is not uncommon as the result of pathologic changes in the optic nerve, etc.); generally it is *partial—i. e.*, one or more of the fundamental colors are not recognized.

According to Helmholtz's theory, three classes of *partial color-blindness* exist—blue-blindness (also called violet-blindness), green-blindness, and red-blindness.

A person afflicted with *blue-blindness* (*yellow-blue blindness,* according to Hering) sees only red and green. He usually confounds blue with green, purple with red, orange with yellow, and violet with yellow-green or gray.

[1] A persistent cramp of the lid, such as occurs in children, unrelieved for weeks at a time, may produce blindness, noticed when the eyes are finally opened, temporary in its character, with normal ophthalmoscopic appearances. In other cases the loss of vision, however, is permanent, with gross changes in the eye-ground. This condition has been referred to under Blepharospasm (page 178).

A person afflicted with *green-blindness* (*red-green blindness*, according to Hering), to quote from Thomson, confounds light green with dark red, does not recognize a dark-green letter on black, but recognizes well a red one on the same background. Preyer states that the most frequent confusions are: brown with dark green, red with green, red with orange, red with yellow, red-yellow with green-yellow, bluish-green with purple.

A person afflicted with *red-blindness* (*red-green blindness*, according to Hering), again to quote from Thomson, confounds light-red colors with dark green, and cannot see a dark-red square on a black ground. According to Preyer, the most frequent confusions are: red with dark green, yellow with green, green with bright red, bluish green with gray, orange with greenish yellow or with red, orange with golden yellow, with grass green, or with red, purple with blue.

Red- and green-blindness are the most usual manifestations of color-blindness; it is often hereditary; the other type—blue-blindness—is not common. According to Lohmann, records concerning the hereditary factors in blue-yellow blindness are wanting. Knies has described congenital violet-blindness; red and purple are not distinguished from each other, both being called red.

In the second variety, or imperfection in the color-sense (reduced color-sense), the individual may correctly recognize brightly marked colors, but confuses colors which are closely allied and the various shades. To him violet and blue and orange and red are difficult distinctions. Dyschromatopsia should be distinguished from partial color-blindness (Landolt).

The *theory of color vision* has been the subject of much speculation, and many theories have been advanced. Two will be mentioned. The *Young-Helmholtz* theory assumes the existence in the retina of three kinds of end-organs, each with its own photochemical substance, which can be decomposed by a certain color; that is, there is a red-sensitive substance, a green-sensitive substance, and a blue-sensitive substance. If a light mainly stimulates the red-, green-, or blue-sensitive substance it gives rise to the sensation, respectively, of red, green, and blue, while simultaneous stimulation of two or more of these substances gives rise to other color-sensations, including white light. A color-blind person, according to this theory, is one in whom two of these substances have a like composition. The *Hering theory* assumes the existence in the retina of a white-black, red-green, and yellow-blue visual substance, which may be either decomposed (disassimilated) or restored (assimilated) by the light. A destructive process, or one of *disassimilation*, in the white-black substance by white light or any other simple or mixed color, produces a sensation of white; a process of restitution, or *assimilation*, in this substance produces the sensation of black. Red light produces disassimilation in the red-green substance, and thus the sensation of red; green light causes a process of restitution, or assimilation, in the red-green substance, and thus the sensation of green. From decomposition of the yellow-blue substance by yellow light arises the sensation

of yellow, while the sensation of blue is produced by a process of assimilation in the same substance. A color-blind person, according to this theory, is one in whose retina the red-green or blue-yellow substance is absent.

Treatment is ordinarily unavailing, but recent investigations indicate that if the defect is ascertained early enough systematic training may succeed in developing the deficient color-sense; hence the importance of the examination of the color-sense in young children.

Congenital Total Color-blindness.—This rare affection has been particularly well studied by Grunert and Uhthoff. To those affected (twice as many males as females) colors appear only as impressions of light and dark. According to Grunert, the colors at the red end of the spectrum seem lighter than to the normal eye, while those at the violet end seem darker. Total color-blindness is nearly always associated with defective central vision, nystagmus, and photophobia. The eye-ground may be normal; or there may be pallor of the disk and macular changes. A central scotoma is common. The eyes are more frequently myopic than hyperopic. Several members of the same family may be affected, and in some instances consanguinity of the parents has been determined.

Congenital Word-blindness.—In this condition the memory for the optic impression of words and letters is greatly deficient or wanting. The affection is more frequent in boys than in girls, although girls are by no means exempt, as has sometimes been stated. As C. J. Thomas points out, it may assume a family type, and in a number of instances more than one member of a family has suffered. Examination reveals normal eyes, good vision after any refractive errors have been corrected, and either inability to learn to spell and to read, or else great difficulty in these respects. Sometimes figures are more readily recognized than letters. In other respects the subjects of this affection are normal and other forms of memory are good, indeed, not infrequently the auditory memory is more developed than in a normal child. The condition is probably due to a congenital defect in the visual memory center for words and letters. As Hinshelwood (to whom we are particularly indebted for early studies of this affection) and all those who have written on the subject since insist, great care should be exercised to detect this affection early in life, because it is much more common than is generally supposed. Its frequency is greater, in all probability, among the lower classes. Much can be done by systematic training—for example, with block letters—as Hinshelwood suggests, so that the child may assist the visual memory by the sense of touch. C. J. Thomas, discussing the treatment of this affection, suggests the phonic method as a suitable one to employ, because in it, at first at least, the visual word-images are ignored.

Reflex Amblyopia.—Certain cases of partial or complete loss of vision have been vaguely attributed to irritations in distant portions of the body—for instance, the presence of parasites in the intestinal canal. In many of these instances, however, a proper investigation has shown that other causes have been active in producing the defective sight.

A number of cases are on record in which an irritation through the branches of the fifth nerve has been supposed to produce an amblyopia, chiefly with disease of the teeth. At all events, in any case of amblyopia unattended with ophthalmoscopic changes, and not readily classified in any of the well-recognized groups, a thorough examination of the teeth is advisable.

Traumatic Amblyopia.—This may occur after severe injuries to the head, especially in the occipital region and the region of the external angular process of the frontal bone; bruises along the course of the spinal cord after a railroad injury; and blows upon the brow in the region of the supra-orbital nerve.

Fig. 222.—Hematoma of optic nerve-sheath. (Patient in the University Hospital.)

In some of the cases there is a fracture across the optic canal, a hemorrhage into the intracranial cavity, or some disorganization of the brain-contents, followed by secondary changes in the optic nerve. (See also page 492.) In other instances no ophthalmoscopic changes are discovered, and the defective vision may be temporary in character, or there may be effusion or hemorrhage into the intersheath of the optic nerve, edema of the retina, and neuritis. *Hemorrhage into the sheath of the optic nerve* after fracture or traumatism of the skull has been observed a number of times and has been especially well studied by Uhthoff. In a specimen recently examined by the author and T. B. Holloway the sheaths were fully distended with blood and there were also many retinal hemorrhages. An extensive fracture of the base of the skull had occurred and had caused this *hematoma of the optic nerve-sheath.*

Amblyopia after railroad injuries is often exaggerated by patients in the hope of securing damages. Recently many soldiers suffering from *shell-shock* caused by their proximity to bursting shells have been examined. The symptoms, according to C. S. Myers, are sleeplessness, amblyopia, contracted visual fields, slight deafness, and sometimes imperfect sense of smell and taste. Parsons suggests that the loss of vision has a definite neural basis associated with what is called a "functional condition."

Amblyopia and amaurosis occur under the influence of disease and the toxic action of certain drugs, due either to a direct effect upon the retina and optic nerve, to an influence upon the visual centers, or to some change, perhaps of vasomotor origin, affecting the blood-supply of these structures.

In this category may be noticed:

1. **Uremic Amblyopia, or Amaurosis.**—This may occur in any form of renal disease, but is more common in the acute nephritis of the eruptive fevers, especially scarlet fever, and of pregnancy than in other varieties of kidney affections. In scarlet fever it appears with albuminuria in the stage of desquamation, and is bilateral, the blindness in many cases being absolute and often associated with brain symptoms: convulsions, vomiting, stupor, coma, and hemiplegia. In spite of the blindness, the preservation of the pupillary reactions is the rule; sometimes the pupils are dilated and motionless.

The ophthalmoscope picture may be negative, or there is a slight neuritis, a little woolliness of the surface of the optic disk or delicate edema of the retina. The *prognosis*, as far as vision is concerned, is good.

The *treatment* does not differ from that which is applicable to the disease which produced it.

2. **Glycosuric Amblyopia.**—In addition to the affections of vision already described in connection with diabetes (paresis of accommodation, premature presbyopia, alterations in refraction, cataract, and retinal hemorrhages), there occurs an amblyopia in this disease in which the visual field is sometimes peripherally intact, sometimes peripherally restricted, and occasionally hemianopic, but in which there is a central color scotoma. Rönne ascribes diabetic amblyopia not to an interstitial neuritis, but to a process of degeneration. This amblyopia may be the only symptom of diabetes, and in any unexplained case of amblyopia the urine should be examined for sugar, a practice which is necessary if color scotomas are found, even if the history of the abuse of tobacco is obtainable.

The prognosis is unfavorable, and the treatment, which should include the usual measures suited to diabetics, is not very efficacious.

3. **Malarial Amblyopia.**—In addition to amblyopia in malarial cachexia with lesions apparent in the fundus, are those cases, without such lesions, due to a special action of the malarial poison upon the optic nerve and the retina. There is transient loss of vision, or complete blindness, lasting from several hours to some days or even months. In most of the instances ophthalmoscopic findings are negative, or the

descriptions are couched in vague terms applied to the retina and optic nerve—"congestion," "hyperemia," and "redder than normal." The affection may be unilateral or bilateral.

4. **Amblyopia from Loss of Blood.**—Loss of sight often follows hemorrhage, more frequently if this is spontaneous than if it is traumatic, and is said to be most complete after hemorrhage from the stomach. It also may follow epistaxis, hemoptysis, urethral and intestinal hemorrhage.

Two very different results may ensue: Either a temporary blindness, owing to the impoverished blood-supply of the visual centers or retina, or a permanent loss of sight and atrophy of the optic nerve. Ward Holden has shown that the amblyopia following hemorrhage is due to degeneration of the retinal ganglion-cells, together with their long processes, which make up the centripetal fibers of the optic nerve.

The ophthalmoscopic appearances vary from a slight pallor to complete atrophic whiteness of the papilla, with contraction of the arteries. The lesions in the unfavorable cases usually do not appear until a week or more after the hemorrhage has taken place. Neuritis and hemorrhages into the retina may also arise. The prognosis is most favorable in uterine cases.

The *treatment* consists in the use of iron, arsenic, and strychnin, complete rest, and an easily assimilated diet. Intravenous saline injections are also recommended as remedial agents (Elschnig).[1]

Amblyopia from the Abuse of Drugs.—A certain number of toxic agents (lead, tobacco, alcohol, etc.) produce an axial neuritis or a degeneration and destruction of the retinal ganglion-cells, with great loss of vision, and these have been described under the general term *orbital optic neuritis* (see page 488).

Amblyopia, more or less complete, may also arise under the toxic influence of nitrate of silver, chlorate of potassium, mercury, arsenic, atoxyl, bisulphid of carbon, nitrobenzol, salicylic acid, oil of wintergreen, cannabis indica, coffee, tea, stramonium, male fern, iodoform, osmic acid, chloral, antipyrin, and lead. The last agent may produce a neuritis, but also an amblyopia without ophthalmoscopic changes. It is usually transient, occurs in acute cases, and has been compared by Gowers to the temporary amaurosis of uremia.

Some of these toxic agents may cause, in addition to the loss of vision, a central scotoma—for example, bisulphid of carbon, stramonium, and iodoform; complete blindness and atrophy of the optic nerve may be the result of the action of others—for instance, male fern and iodoform.

The loss of vision which occurs under the influence of three substances—quinin, methyl-alcohol, and atoxyl—deserves special mention.

Quinin Amaurosis.—Although in most instances *quinin amblyopia*, or *amaurosis*, follows the ingestion of a large quantity of the drug, occa-

[1] Sudden blindness with preserved pupillary reaction and without ophthalmoscopic changes has been noted in whooping-cough, and is probably due to edema between the corpora quadrigemina and occipital lobes. Sudden blindness in old persons with arteriosclerosis has been observed (Uhthoff).

sionally the symptoms are caused by moderate doses. The author has seen 12 grains (0.78 gm.) produce decided temporary amblyopia in a susceptible and neurotic woman.

The characteristic clinical features of quinin amaurosis are total blindness subsequent to taking large doses of the drug, extreme pallor of the optic disks, marked diminution of the retinal blood-vessels in number and caliber, and contraction of the field of vision. Other symptoms which have been noted are: diminution of the color- and light-sense, dilated pupils, and immobile iris during the blind stage, and occasionally anesthesia of the cornea. Usually the effect of quinin upon the ear is manifested by deafness and tinnitus.

The restoration of central vision may be perfect or incomplete. The contracted field of vision gradually widens out, but does not regain its normal limits. The disk may remain pallid and quite atrophic looking years after the poisoning; in other instances it resumes its normal tint. In one case (Gruening) a cherry-colored spot was noted in the macula, in another a scotoma in the visual field. Occasionally the blindness is permanent.

The first effect of the toxic influence of quinin is to lessen the blood-supply of the retina and optic nerve, and later, as the author has experimentally shown in dogs, permanent optic-nerve atrophy ensues. Ward Holden has demonstrated, and his results have been fully confirmed by Drualt, Birch-Hirschfeld, and a number of other observers, that the blindness is due to a degeneration of the ganglion-cells and nerve-fibers of the retina, followed by an ascending degeneration of the optic nerve.

The *treatment*, in addition to the discontinuance of the drug, consists in the administration of nitrite of amyl, which will cause temporary improvement in vision, and of the exhibition of strychnin and digitalis.

Methyl-alcohol Blindness, or Amaurosis.—The amount of wood-alcohol which may cause blindness represents a varying quantity. Thus, blindness and atrophy of the disk have followed the ingestion of 2 to 5 drams (7.8–19.4 gm.), while recovery after drinking ½ pint (236 c.c.) of this liquor has been observed (Moulton). In short, methyl-alcohol intoxication is an example of idiosyncrasy (F. Buller, C. A. Wood). The number of immune persons, however, cannot be great. Methyl-alcohol itself, Columbian spirits, other varieties of purified wood-alcohol, and the drug in the form of an adulterant for ethyl-alcohol in cheap whiskies and other alcoholic beverages, as well as in Jamaica ginger, certain essences, bay-rum, cologne water, etc., are capable of producing the most violent general toxemia and visual disturbance. Igersheimer believes the influence of methyl-alcohol depends upon the admixture of fusel oil. Briefly, the symptoms are these: Intense gastro-intestinal disturbance if the dose is not too large, followed, if it is greater, by severe headache, giddiness, and coma; rapid failure of sight, which may improve, but soon relapses; contracted visual fields and usually absolute central scotomas; and, finally, total or nearly total blindness. Oph-

thalmoscopically, there have been noted blurring of the edges of the disk, positive neuritis (rare), and complete atrophy without signs of preceding inflammation. In many instances there is diminution in the size of the retinal vessels. Occasionally, there is decided pain on movement of the eyes or on pressing them backward into the orbit. The *prognosis* of methyl-alcohol poisoning is most unfavorable. A number of fatal cases have been reported. Not only may the poison enter in the usual manner through the stomach, but blindness has resulted from inhalation, aided by absorption, as the author has shown, through the cutaneous surface. A very few examples of restoration to nearly normal vision have been reported. The blindness depends, as Holden and Birch-Hirschfeld have demonstrated, upon nutritive changes in the ganglion-cells of the retina. It is possible that there may be a simultaneous action on the ganglion-cells and the tissues of the optic nerve (Gifford). The treatment of this form of amaurosis recommended by F. Buller and C. A. Wood includes in the early stages pilocarpin and potassium iodid, later strychnin hypodermically and by the mouth.

Arsenic Amblyopia.—Atoxyl has been much employed in the treatment of various conditions, notably certain skin diseases, chlorosis, syphilis, and trypanosomiasis. Serious visual disturbances have followed its use in a number of instances. The following have been reported: Reduction of visual acuteness from one-half to complete blindness; contraction of the visual field, especially on the nasal side; pallor and atrophy of the optic disk, with narrowing of the retinal vessels; usually no central scotoma, but at times a central scotoma for colors and sometimes visual hallucinations and colored vision (cyanopsia). In one patient retinal hemorrhages were found. The amount of the drug which has produced these visual disturbances has varied considerably: 1.2 grams, given subcutaneously for anemia, within twenty-six days (Steinebach); 5.1 grams within twenty-six days (Lesser and Greeff); 50 grams within seven months (Von Krüdener); 4.5 grams within one month (Kopke). Koch observed a number of cases of blindness without ophthalmoscopic change after injection of 1 gram of atoxyl for the cure of sleeping-sickness. The blindness has been ascribed to optic-nerve atrophy with primary involvement of the retina, and to retrobulbar neuritis and consecutive atrophy; it has also been attributed to central lesions.

Blindness following the administration of other *aryolarsenates*, for example, soamin, arsacetin, hectin, orsudan, have been reported. The harmful action is due to the anilin in these compounds and not to the arsenic. Amblyopia due to inorganic arsenic compounds presents features entirely different from those which the organic preparations produce and the prognosis is good (Schirmer). The aryolarsenates cause progressive optic-nerve atrophy. It has been suggested that the primary action of the drug may be on the blood-vessels (E. T. Collins). The harmful dose has varied; much depends on idiosyncrasy. Salvarsan does not cause blindness, *i. e.*, it has no poisonous influence on the healthy optic nerve.

Hysteric Visual Disturbances (*Amblyopia, Amaurosis, Asthenopia*).—Hysteric amaurosis is characterized by complete abeyance of the visual sensation. It occurs both as a unilateral and a bilateral affection, the former being far more frequent than the latter. The subjects of this condition are more frequently females than males. Occasionally, the blindness lasts but a very brief period of time, and occurs during a crisis; at other times it lasts for weeks, months, and, it is said, for years. The eye-grounds are normal. Usually the pupils react to the influence of light. Sometimes only a feeble contraction follows the light stimulus; occasionally the pupils are dilated and insensitive to light (Kerneis). Generally it is possible to prove by ordinary prismatic, stereoscopic, and other tests that the supposed blind eye really sees.

In place of amaurosis, incomplete anesthesia of the visual sense, or hysteric amblyopia, may occur. This includes reduction of visual acuteness, disturbances of the visual field for white and for colors, dischromatopsia, and achromatopsia. The essential characteristic of the visual field in hysteria is concentric contraction, which is evident at the beginning of the examination, and is not produced by repeated measurement (retinal tire field), and the amount of reduction varies from a slight contraction to such extreme restriction that the most peripheral circle is just beyond the fixing-point. Sometimes the field has a *tubular* character—that is, the contracted visual field maintains the same size, no matter at what distance from the examined eye the point of fixation is placed. Similar reductions take place in the field for colors. A somewhat characteristic variation is that the red field is the last to be affected, with the result that its extent may exceed that of blue, and become the most peripheral of the color circles. Occasionally it is the most peripheral circle for the entire field. This is the so-called *inversion of the color-field*. Sometimes there is an excessive extent of the color-circles. These visual field phenomena in hysteria are, according to Babinski, due to suggestion; if this can be eliminated, he maintains they do not develop. It should be remembered that inversion of the color-fields is not peculiar to hysteria; it has been observed in brain tumor, ataxia, hemorrhage in the brain, and in certain toxemias, notably those produced by lead and nitrobenzol. A rare hysteric phenomenon is central scotoma; zonular scotoma and the so-called oscillating field have been observed. *Crossed amblyopia*—that is, complete or partial blindness on the same side as the hemianesthesia, and associated with some deficiency of acuteness of vision upon the opposite side—is sometimes an hysteric manifestation. Hemianopsia in an enduring form is never due to hysteria. As a temporary visual-field phenomenon it has been observed.

Hysteria produces many other functional disturbances of the eye—monocular diplopia, ptosis, blepharospasm, conjugate deviation of the eyes, and the great symptom-group gathered under the term "retinal asthenopia" (see page 423).

The *prognosis* of these cases in the main is good, although the blindness may last for long periods of time.

The *treatment* consists of measures calculated to improve the condition of the patient—massage, rest, electricity, and tonics.

Pretended Amblyopia (*Malingering*).—For the purpose of escaping irksome duties—for example, in the army—or to excite sympathy patients will occasionally pretend to be blind in one eye. In order to detect the deception many plans have been originated. Three methods will be described:

1. **The Diplopia Test.**—This is performed in the same manner as the ordinary examinations of the external ocular muscles with prisms. The subject is seated before a lighted candle at 20 feet distance, and a 7° prism placed before the admittedly sound eye. If, now, superimposed double images are acknowledged, there is binocular vision, and the fraud is detected. The examiner may vary the test by placing the prism before the supposed blind eye, either base up or base down.

2. **Harlan's Test.**—This is an extremely useful and simple test, and is performed as follows: Place an ordinary trial-frame upon the subject's face and put before the admittedly sound eye a high convex glass (+ 16 D), and before the eye which is claimed to be blind a plain glass or a weak concave spheric (− 25 D), which will not interfere with vision. If letters placed at a distance of 6 meters are read, the act of reading must have been done by the eye which was claimed to be sightless, inasmuch as vision at that distance with the other eye is excluded by the presence of the high convex lens. The test may be further elaborated by covering the pretended blind eye and requesting the patient to read the letters; if he is unable to do so, the fraud is at once exposed.

3. **Tests with Colored Glasses and Letters.**—These are numerous. The method generally employed, or some modification of it, is known as *Snellen's method.* The patient is required to look at alternate red and green letters. The admittedly sound eye is covered with a red glass, and if the green letters are read evidence of fraud is present. Instead of a red glass, a green glass may be used, through which the red letters will be invisible. Ingenious letters, based upon the fact that red upon a white background viewed through a red glass disappears, and viewed through a green glass appears black, have been designed. Tests with stereoscopes may also be made to detect malingering.

If a malingerer claims to be blind in both eyes, these tests will not avail, and he can be detected by placing a careful watch over him. The fact that the pupil contracts on exposure to light does not prove that there is sight in the eye, because, as Swanzy pointed out, a lesion in the center of vision, or in the course of the fibers connecting this center with the corpora quadrigemina, producing absolute blindness, would still permit a perfect reaction of the pupil to light. Priestley Smith and E. Jackson suggest the following test for feigned *binocular blindness.* Place a lighted candle in front of the subject; now hold a 6°

prism, base out, before one eye; if both eyes see, the one behind the prism will move inward, and on removing the prism will move outward, the other eye remaining fixed.

Night-blindness (*Functional Night-blindness; often incorrectly termed Hemeralopia, but properly Nyctalopia*).—It has already been pointed out that night-blindness is one of the early symptoms of pigmentary degeneration of the retina. In the present condition, however, there are no retinal lesions.

It is a functional complaint, consisting in a diminished sensibility or imperfect adaptation power of the retina (Treitel), due, apparently, to exposure of the eye to strong light and glare, together with a debilitated and often scorbutic state of the system. It affects residents in tropical countries, often soldiers and sailors, and has been occasionally observed in large schools, usually in the early spring or summer (Nettleship, Snell). It has been reported as an endemic in certain countries, especially in Russia during the Lenten fasts. Other causes are diet deficient in fat and albumin, disease of the liver, malaria, and alcoholism.

Krienes divides the affection into *acute essential nyctalopia* (hemeralopia) and *chronic nyctalopia*, and he gives the following syllabus of symptoms: Decided dread of light, abnormal width of the pupil in the dark, depreciation of the central quantitative color-sense, particularly the blue sense in daylight, narrowing of the color-fields in daylight, particularly the blue field, abnormal shrinking of the visual field for white and colors in increasing twilight. Other not absolutely constant symptoms are loss of visual acuteness by daylight, shrinking of visual field for white in daylight, retinal tire field, paresis of accommodation, epithelial xerosis, erythropsia, and xanthopsia (see also page 230).

Night-blindness is occasionally a family disorder. Bordley has described a negro family of night-blind persons extending over five generations. The subjects eventually became blind, and shortly after blindness death ensued. Nettleship has published a history of stationary night-blindness in nine consecutive generations.

Treatment.—This includes the administration of iron, quinin, strychnin, and cod-liver oil, according to the indications. Dark-colored glasses should be worn. If scurvy is present, the diet and remedies suited to this condition should be prescribed.

Day-blindness (*Often incorrectly termed Nyctalopia, but properly named Hemeralopia*).—This is an affection, or rather a symptom, as the name implies, characterized by the fact that its subjects see better on dull days and in the dark than in a bright light. The visual field is not concentrically contracted.

This symptom occurs with the condition described by Arlt as *retinitis nyctalopia*, and with orbital optic neuritis of the chronic type (tobacco amblyopia, see page 490). It also occurs in other affections of the optic nerve and in some diseases of the retina. The same condition may be present in certain congenital anomalies—albinism, coloboma of the

iris, and irideremia. It also occurs as an idiopathic affection, and may develop in those who have long been excluded from the light. It may be congenital, and may be associated with an amblyopia of like origin.

A tonic *treatment* should be tried and the retina gradually educated to sustain bright light.

Snow-blindness.—As this ordinarily is seen in northern regions, it is an affection of the conjunctiva. There are burning pain, photophobia, blepharospasm, hyperemia of the conjunctiva, and chemosis. In severe cases there may be ulceration of the cornea. The pupils are small, and there is congestion of the retina. The visual acuteness may be unaffected, or it may be distinctly lessened, especially if corneal complications arise; under these conditions restricted visual fields, both day- and night-blindness, red-green blindness (Lohmann), and scotomas have been observed. The local irritation in snow-blindness is analogous to sunburn, and like it is due to the action of ultraviolet rays. The pain caused by *glare* is due to excessive contraction of the orbicularis and to pressure of the supra-orbital nerve against the frontal bone.

Symptoms analogous to so-called "snow-blindness" develop as the result of exposure of unprotected eyes to any type of light rich in ultraviolet—for example, a naked arc light (*electric ophthalmia*); molten metal in electric welding; the flash of a short circuit high-tension current, and a mercury vapor lamp. To the *injurious effect of light* in these circumstances, interpreted by the irritative phenomena already described, Parsons has applied the term *photophthalmia*. Although the ultraviolet rays probably cause the greater damage, rays of greater wave length—infra-red and luminous rays—are also capable of harmful action. For a long time after subsidence of photophthalmia there may be persistent *asthenopia*, ciliary pain, frontal and other placed headaches. Those who are much engaged in work with Röntgen rays often suffer from decided conjunctival hyperemia or positive conjunctivitis (*x-ray conjunctivitis*).

Experimentally, it has been demonstrated that ultraviolet rays may cause changes in the lens (Hess) and in the retina (Birsch-Hirschfeld). These changes are not likely to occur under ordinary conditions in healthy persons. Special forms of glass supposed to have the power of absorbing ultraviolet rays have been devised; all of them have a greenish-yellow tint, chief among them are "Enixanthos," Fieuzal glass, and "Euphos" glass. According to Parsons, almost colorless didymium glass is the best protection against ultraviolet rays.[1]

Erythropsia, or Red Vision.—Colored vision in glaucoma (iridescent vision), in the form of variously tinted halos about the lamplights, has been described, and patients with blind eyes occasionally complain of being conscious of colored lights, owing probably to some irritation of the visual centers.

[1] "Some Effects of Bright Light on the Eyes," by J. Herbert Parsons, Trans. of Section of Ophthalmology, Amer. Med. Assoc., 1910.

Erythropsia in most instances has been noted after cataract extraction. Visual acuteness is not affected, but everything appears of a red or violet color. According to Fuchs, erythropsia can be caused by the visible rays alone, but a simultaneous action of the ultraviolet rays is probable. An uncommon phenomenon is *blue vision* or *kyanopsia.* Bromid of potassium is indicated, and is said to ameliorate these symptoms. *Green vision* has been noted after cataract extraction and corneal wounds, and in connection with diseases of the optic nerve and retina—for example, with tabetic optic-nerve atrophy (H. W. Dodd).

Micropsia and **macropsia** have been described in connection with syphilitic retinitis. They may appear as functional disorders in hysteric cases.

CHAPTER XVIII

AMBLYOPIA OF THE VISUAL FIELD, SCOTOMAS, AND HEMIANOPSIA

The importance of perimetric measurements in the study of various forms of ocular disease, especially in glaucoma and in affections of the retina, choroid, and optic nerve, has been noted (for the methods of examination consult Chapter II). There remain to be considered certain conditions in which a defect in the field of vision constitutes one of the most prominent symptoms.

1. **Partial Fugacious Amaurosis** (*Flimmer-scotom; Migraine Ophthalmique*).—The symptoms are: A sense of vertigo; a positive darkening of the field of vision of each eye, beginning at the center and widening out in a vibratory movement until it overspreads the field, with corresponding sinking of the central acuteness of sight; or a scotoma surrounded by flashing zigzag lines, and cessation of the amaurosis with the onset of headache and vomiting; a true hemianopia without light or flashing is also described. These symptoms may be, and often are, a prodrome of hemicrania or migraine, but they also arise without it, and may occur in syphilitic subjects. The condition probably depends upon circulatory disturbances in the occipital lobes, or upon vascular spasm.

The *treatment* is directed toward the headache, the partial amaurosis being exceedingly temporary in character, and includes the measures suited to migraine. Syphilis calls for the usual remedies.

2. **Amblyopia of the Visual Field** (*Anæsthesia Retinæ*).—This functional disturbance as part of a general neurosis has been described on page 423. Because of the peculiar changes in the visual field many authors prefer the name "amblyopia of the visual field" to that of "anesthesia of the retina."

Fatigue restrictions of the visual field, in the form already described, are seen after injuries (*traumatic neurosis*), and sometimes with *traumatic anesthesia of the retina*. The element of hysteria cannot always be eliminated, and the phenomena described in connection with hysteric ocular manifestations may predominate.

3. **Scotomas.**—Any lesion which blots out the function of a portion of the retina produces a corresponding blind area in the field of vision, or a scotoma—for example, a hemorrhage, a patch of retinochoroiditis in the macular region, or spots of disseminated choroiditis in the periphery of the eye-ground. In rare instances the scotoma seen by a patient with central retinochoroiditis is colored. Papillitis causes an enlargement of the natural blind-spot, and retrobulbar neuritis a central scotoma. The different forms which scotomas assume are

described on page 85. The scotomas associated with chronic glaucoma are depicted on page 373. Unilateral scotomas may occur in hysteria, in neurasthenia (central exhaustion scotomas), with menstrual disorders, in obstruction of the central artery of the retina, and with disease of the macular cortical center. *Ring-shaped* or *annular* scotomas have attracted much attention. Ordinarily, they are to be explained by the presence of chorioretinitis. According to Burnett, circumscribed neuritis of the bundles of the optic nerve supplying the intermediate retinal zone may explain some cases. Such scotomas may be caused by chorioretinitis, pigmentary degeneration of the retina, and by interstitial neuritis, choked disk, sinusitis (ethmoid and sphenoid disease), and glaucomatous atrophy of the optic nerve (see page 374). They have also been observed in hysteric subjects and have been reported as part of the symptomatology of migraine (Zentmayer).

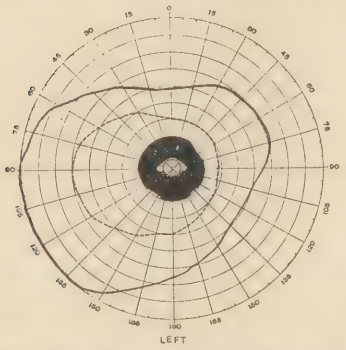


Fig. 223.—Ring-shaped scotoma from a case of interstitial neuritis.

In addition to these diseases certain affections of the optic nerve are accompanied by a scotoma.

Following the classification of Jensen, these may be described as:[1]

(*a*) **Central Amblyopia with Scotoma** (*Toxic Amblyopia*).—This affection has been described on page 490.

(*b*) **Chiasmal Central Amblyopia.**—Central scotomas, difficult to distinguish from those occurring in toxic amblyopia, are sometimes the initial signs of chiasm disease. According to Nettleship, the loss of the central field in the earlier stages is more abruptly defined than in tobacco amblyopia. With increase in the size of the growth they may expand into complete bitemporal hemianopsia (see page 515). As the visual phenomena in pituitary body disease have assumed such importance in ophthalmic and neurologic examinations, a brief résumé of them follows:

Visual Phenomena in Pituitary Body Disease.—These consist in impairment of vision, varying from blurred sight to complete blindness; intra-ocular optic-nerve alterations, varying from partial or general pallor of the nerve-head to partial or complete atrophy, less frequently postneuritic atrophy, choked disk, and non-prominent optic neuritis; alterations in the visual fields for form and colors, especially homonymous and heteronymous hemianopsia and scotomas. Other ocular symptoms which have been described are visual hallucinations, chromatopsia, especially cyanopsia, persistent photophobia, palsy of various exterior ocular muscles, nystagmus, exophthalmos, thickening and pigmentation

[1] A translation by Dr. G. A. Berry of a lengthy abstract of Jensen's article on "Diseases of the Eye Accompanied by a Central Scotoma" appeared in the Ophthalmic Review, January, 1891.

of the eyelids with hypertrophy of the palpebral glands. A very early symptom, noticeable sometimes for weeks and months before fundus changes are detected, is a form of blurred vision, characterized by ill-defined indistinctness of sight. Visual disturbances are more frequent in primary hypopituitarism than in acromegaly. Generally the ophthalmoscopic appearances are those of so-called simple atrophy; choked disk is rare. Even when the pallor of the disk gives every indication of simple atrophy, marked improvement in vision may follow successful operation or prolonged organotherapy. Evidently there is a physiologic block to light impulses, and not at this period a destruction of the nerve-fibers. While bitemporal hemianopsia is a common visual field defect, homonymous lateral hemianopsia is not infrequent, especially if with homonymous defects are included what Cushing calls the tendencies in this direction. In all cases the defect for colors precedes that for form. There may be unilateral hemianopsia. In bitemporal hemianopsia in this disease it will be found that the temporal field tends to be lost from above downward. This is the so-called "temporal slant." A peculiarity of the visual fields in pituitary body disease is the variations which they undergo, hence the necessity of frequent perimetric observations.

Central and paracentral scotomas are common, especially the paracentral varieties; they may be situated to the outer side of each fixation-point, that is, they are bitemporal hemianopic scotomas. These scotomas antedate the more elaborate visual field defects and they may expand into complete hemianopsia. Sometimes the defect may be detected upward and outward, forming a quadrant. The most elaborate and satisfactory study of the visual field phenomena in pituitary body disease has been made by Harvey Cushing and C. B. Walker, and they have demonstrated the necessity of frequent examination of the visual fields with test-objects of various sizes. (See footnote, page 82.)

(*c*) **Stationary Optic Atrophy with Scotoma.**—This is characterized by a scotoma similar to the one which occurs in toxic amblyopia, but much more decided. There are marked diminution of central vision, a depreciation of the color-sense, and ophthalmoscopically the appearances of optic-nerve atrophy. The process is stationary, and vision does not improve under treatment. Jensen finds this affection exclusively in men before their thirty-fourth year. It has a hereditary tendency, and is said to be caused by exhaustion and lack of sleep. Sometimes no cause can be demonstrated. Preceding the atrophy there may be slight neuritis.

In the cases of *hereditary atrophy* of the optic nerve recorded by W. F. Norris, the ophthalmoscopic changes commenced with a stage of cloudy swelling of the disk and passed on to a gradual death of the nerve. The disease began with a central scotoma, first for color, but gradually this became complete. Both the males and females of the family were affected (see also page 486).

(*d*) **Progressive Optic Atrophy with Scotoma.**—This includes the class of cases in which the optic-nerve atrophy of spinal disease (tabes dorsalis and disseminated sclerosis) is associated with a scotoma. The

scotoma is central and shaped like the one in tobacco amblyopia, but as the disease progresses the peripheral field contracts, and finally it becomes difficult to detect the central defect. A central scotoma in tabetic atrophy of the optic nerve, according to Fuchs, is of more frequent occurrence than the ordinary records would seem to indicate. The scotoma is nearly always bilateral, and he regards it not as an accidental complication, but as an integral part of the tabetic process. Some authors maintain that the influence of tobacco and alcohol in the formation of these scotomas cannot be eliminated. Central scotoma is common in insular sclerosis.

(*e*) **Optic Neuritis with Scotoma.**—An unusual symptom of intraocular neuritis caused by meningitis is a central scotoma, either relative or absolute. The student should not confuse this with an enlargement of the natural blind-spot due to the inflammatory swelling of the nerve-head. Ring scotoma has been reported with choked disk caused by brain tumor, and in optic neuritis due to sinusitis a central scotoma may develop.

As has already been pointed out, the cause of central scotoma in orbital optic neuritis (toxic amblyopia) is degeneration of the papillomacular bundle in the optic nerve or a destruction of the macular ganglion-cells. Whether a partial affection of the optic nerve will explain all cases of central scotoma remains to be seen, and Jensen suggests that a common central cause may be active.

Obscuration of One-half of the Visual Field, or Hemianopsia.[1] —In diseases of the eye—*e. g.*, glaucoma—one-half of the visual field may be wanting, and also in optic-nerve atrophy and neuritis, even if they are unconnected with disease of the visual pathway. These cases, however, are not included in the present account.

Hemianopsia is that defect of vision characterized by an obscuration, usually in each eye, of one-half of the field, which occurs under the influence of a lesion situated at the optic chiasm, in the optic tract, in the visual radiations, or at their ultimate destination in the brain (occipital lobe).

Visual Tract.—The visual tract, or visual conduction paths, may briefly be described as follows:

The retina is a highly evolved structure, which, from the histologic standpoint, may be divided into three layers: (1) The *layer of the neuro-epithelium*, composed of two strata, namely, the *layer of rods and cones* and the *external nuclear layer*, the former constituting the specialized outer portions and the latter the nucleated bodies of the *visual cells;* (2) the *layer of the bipolar cells*, which by some authorities are looked upon as the peripheral visual neurons; (3) the layer of the ganglion-cells.

The long processes, or axons, of the ganglion-cells pass into the *nerve-fiber layer of the retina*, reaching the *papilla* or *nerve-head*, and proceed to the *optic nerve*. Having reached the *optic chiasm*, a portion of the fibers of one optic nerve cross over and enter the *optic tract* of the opposite side, forming the *crossed fasciculus*, while a certain number of other fibers do not cross, but enter the optic tract of the same side, forming the *non-crossed fasciculus*. The non-crossed fasciculus arises chiefly from the tem-

[1] The terms *hemiopia* and *hemianopsia* are often used synonymously. Really, hemiopia signifies loss in the perceptive power of one-half of the retina, while hemianopsia means obscuration of one-half of the visual field (Seguin). Other names which are used are hemianopia and hemiablepsia.

poral side of the retina, while the crossed fasciculus arises from the ganglion-cells of the nasal side of the retina. The bundle from the macula lutea, called the *macular fasciculus*, or *papillomacular bundle*, in general terms, is situated in the central part of the optic nerve and maintains its central position in the optic chiasm and in the optic tract, and is composed of crossing and direct fibers. The optic tract on each side behind the chiasm passes around the cerebral peduncle of the same side and arrives at the junction of the interbrain and midbrain, and divides into a lateral and a medial root. The fibers of the lateral root terminate in the *lateral geniculate body*, in the *pulvinar* of the *thalamus*, and in the *superior colliculus of the corpora quadrigemina*. These structures have been designated the *primary visual ganglia* or *primary optic centers*.[1] The corpora quadrigemina are not concerned in the act of vision, but in the activity of the pupil. The medial root of the optic tract has no connection either with the retina or with the optic centers of the interbrain and midbrain.

From the regions just described fibers proceed backward through the posterior part of the *internal capsule* to the cortex. These are the *optic* or *visual radiations*, or *fibers of Gratiolet* or *of Wernicke*. Passing through the internal capsule, they cross the sensitive fibers coming down from the hemisphere, and spreading out like a fan, rise upward, wind outside of the tip of the lateral ventricle, to reach their destination at the lower part of the median surface of the *occipital lobe*—that is, the cortical termination of the visual tracts. The exact area occupied by the cortical center of vision has not been determined. In general terms it is situated about the *cuneus* and *calcarine fissure*, and does not comprise the whole of the occipital lobe.

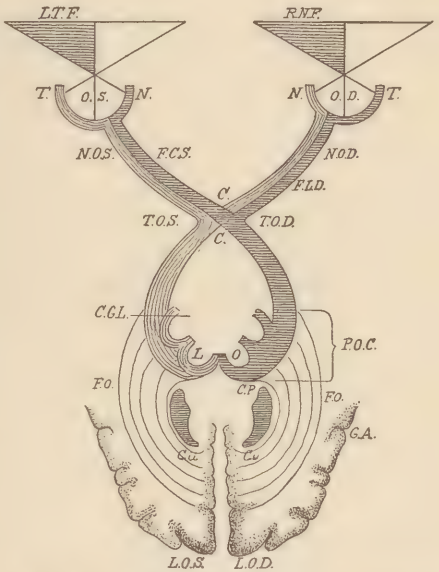


Fig. 224.—Diagram illustrating the visual path and its relation to the visual field, left lateral hemianopsia being shown (Seguin).

By comparing the description of the varieties of hemianopsia which follow with Fig. 224, the student will understand the mechanism of their development.

The following from Seguin explains the lettering of the illustration:

L. T. F., left temporal half-field; *R. N. F.*, right nasal half-field; *O. S.*, left eye; *O. D.*, right eye; *N.*, nasal, and *T.*, temporal halves of the retinas; *N. O. S.*, left optic nerve; *N. O. D.*, right optic nerve; *F. C. S.*, left crossed fasciculus; *F. L. D.*, right lateral or non-crossed fasciculus; *C.*, Chiasm or decussation of the fasciculi; *T. O. D.*, right optic tract; *T. O. S.*, left optic tract; *C. G. L.*, corpus geniculatum laterale (medial corpus geniculatum and its arms are omitted); *L. O.*, optic lobes (corpora quadrigemina); *P. O. C.*, primary optic centers (including corpora quadrigemina, corpora geniculata, and pulvinar of the optic thalamus); *F. O.*, optic fas-

[1] According to W. G. Spiller, the chief "primary" optic center is the external geniculate body. The pulvinar of the optic thalamus is also an important "primary" optic center. The anterior colliculus of the quadrigeminal body in man has an unimportant relation to vision. The hypothalmic body, the habenula, the internal geniculate body, probably are not part of the visual system (consult Spiller, "A Case of Complete Absence of the Visual System in an Adult," Univ. of Penna. Medical Bulletin, February, 1902).

ciculus, radiating visual fibers of Gratiolet in the internal capsule; *C. P.*, posterior horn of the lateral ventricle; *G. A.*, region of the gyrus angularis; *L. O. S.*, left occipital lobe; *L. O. D.*, right occipital lobe; *Cu.*, cuneus and subjacent gyri constituting the cortical visual center in man. The shaded lines represent the parts connected with the right halves of the retinas.

Varieties of Hemianopsia.—Hemianopsia is divided into *horizontal*, in which the dividing-line between the darkened and preserved field is horizontal; and *vertical*, in which the dividing-line is vertical.

1. *Horizontal hemianopsia* (altitudinal) may be inferior or superior, both lower or both upper half-fields being wanting. In addition to diseases of the eye, it is possible that such a condition could arise under the influence of a lesion so situated as to press upon the upper or lower part of the chiasm, or downward upon one optic tract, or upon the lower or upper part of both optic nerves. A double lesion in front of the chiasm may produce loss of the upper half of the field in one eye and of the lower half of the field in the other eye.

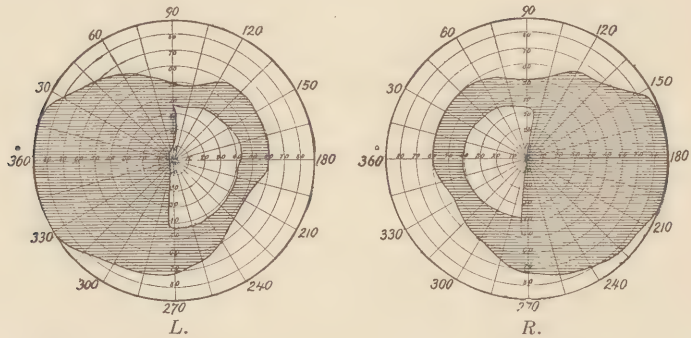


Fig. 225.—Bitemporal hemianopsia. The shaded areas represent the portions of the fields which are dark, and it is evident that there are entire loss of both temporal fields and some contraction of the preserved fields (from a case of acromegaly).

2. *Vertical Hemianopsia.*—This is subdivided into several varieties:

(*a*) *Bitemporal hemianopsia* (peripheral), in which both temporal fields are wanting, is characteristic of lesion of the chiasm. The defect is not necessarily complete from the beginning. Color-sense at first may be alone affected, followed later by loss of form- and light-sense. In place of complete bitemporal hemianopsia there may be paracentral or bitemporal hemiopic scotomas, which gradually broaden into bitemporal hemianopsia (see also pages 510 and 511). There are a number of types or combinations of bitemporal visual field defects. Wildbrand described nine, Cushing and Walker have elaborated his list.

(*b*) *Binasal hemianopsia*, in which both nasal fields are wanting, is rare. Unilateral nasal hemianopsia also occurs.

(*c*) *Homonymous hemianopsia* (central), in which the corresponding half of the field in each eye is wanting: thus, both right or both left fields are darkened, in the former case indicating loss of function of the left half of each retina and designated *right homonymous lateral hemian-*

opsia, and in the latter case indicating loss of function of the right half of each retina, and designated *left homonymous lateral hemianopsia* (see Figs. 226 and 227).

This is the commonest form of hemianopsia.

Hemianopsia may be *complete—i. e.*, the entire half of each field is wanting—or *incomplete*, *i. e.*, a portion of each half-field is wanting, the

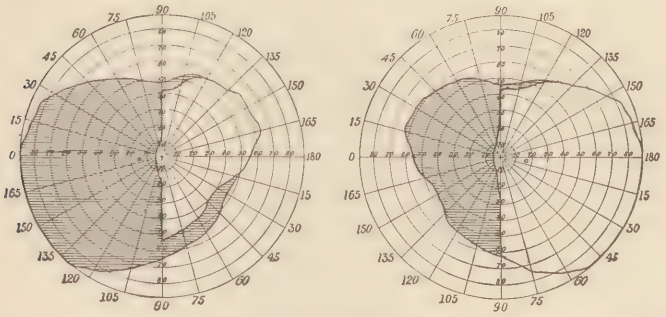


Fig. 226.—Left homonymous hemianopsia, from a case of gunshot wound, with suspected lesion of the right cuneus. The shading shows where vision was lost (from a patient under the care of the late Dr. S. Weir Mitchell in the Infirmary for Nervous Diseases).

defect usually being in the form of a quadrant (Fig. 227). The preserved half-fields may be of their normal size, or they may exhibit concentric contraction.

Finally, the hemianopsia may be *absolute—i. e.*, all the three functions of sight (perception of light, of form, and of color) are wanting—or it may be *relative*, *i. e.*, perception of color only is lacking, light-sense

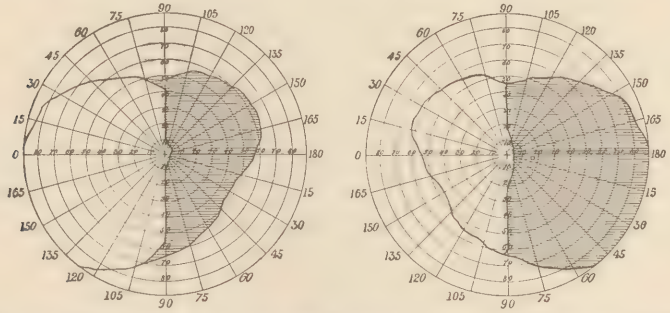


Fig. 227.—Right homonymous hemianopsia (from a patient under the care of the late Dr. Wharton Sinkler).

and form-sense being preserved; or perception of color and form is wanting in the deficient area of the field, but the light-sense is preserved. Those cases in which the half-defect is present for colors alone are described under the name *homonymous hemiachromatopsia*. They probably represent a cortical lesion of less intensity than one which produces absolute hemianopsia. In a remarkable case of this kind which the author

has studied with Dr. J. William White, at the onset the hemianopsia was absolute; later light-sense and form-sense returned. The obliteration of the color-sense remains, although in all other respects the patient has recovered. Non-cortical lesions may also produce hemiachromatopsia.

Peculiarities of the Dividing-line.—The dividing-line may exactly cut the fixing-point, or, as is usual, it may pass around this point and leave it within the region of preserved vision. The want of uniformity between the seeing and the blind areas may be manifested by the failure of the dividing-line to coincide with the vertical meridian for some distance, by its assuming an oblique or irregular direction, or by forming an open angle. A number of theories have been advanced to account for these peculiarities. They have been explained by Schmidt-Rimpler by assuming that there are anastomoses of the fibers from each optic tract in the retinas as well as in the optic nerves and chiasm.

A number of cases of *double* homonymous hemianopsia are recorded, in which there was preserved a small central field of each eye. This

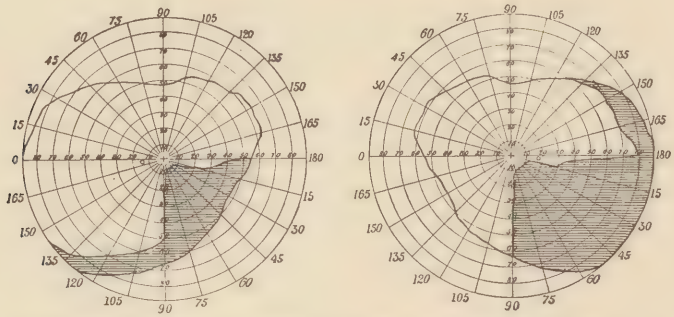


Fig. 228.—Quadrant homonymous anopsia or tetranopsia, shading as before. A quadrant of each field is wanting. The lesion is probably in the cuneus.

indicates that there is a region in the cortical visual centers which supplies the macula lutea and that this has not been destroyed.

Significance of Hemianopsia.—Typical bitemporal hemianopsia of permanent character is caused by a lesion—tumor, aneurysm, exostosis, arterial disease, etc.—which destroys the conductivity of both crossed fasciculi, leaving the non-crossed fasciculi unaffected (see page 515). It is a common symptom of pituitary body disease (neoplasm or struma). Affections of this region, however, may cause other varieties of visual field defects, concentric contraction, homonymous hemianopsia, and scotomas.

A true chiasmal variety of binasal hemianopsia probably does not occur, although the visual field defect has been attributed to disease of the lateral angles of the chiasm. Most of the cases seem best explained by a bilateral inflammation of the trunks of the optic nerves in front of the chiasm. Unilateral hemianopsia, if not caused by disease within the eye, could originate from injury or lesion in one optic nerve. A nasal

hemianopsia on one side could be produced by a lesion affecting the lateral portion of the chiasm involving the non-crossing fibers of one eye.

Homonymous lateral hemianopsia is caused by a lesion situated in the occipital lobe, the optic radiations, the internal capsule, the primary optic centers, or the optic tract (see Fig. 224).

(*a*) The lesion in hemianopsia is on the opposite side of the dark fields.

(*b*) If the preserved fields are accompanied by concentric contraction, the smaller half-field will be in the eye opposite to the lesion; contraction of the preserved half-field is most common with lesions of the cortex, but also may occur in lesions of the tractus.

(*c*) If the hemianopsia is relative, the lesion is probably in the cortex; but cortical lesions are not excluded by absolute hemianopsia.

(*d*) A lesion confined to the cuneus, or to it and the gray matter immediately surrounding it, on the mesial surface of the occipital lobe, produces homonymous lateral hemianopsia without motor or sensory symptoms, at least without these as a direct consequence of the lesion, although they may appear as *indirect*, or, as they are sometimes called, *distant symptoms*.

(*e*) A lesion producing typical hemiplegia, aphasia, if the right side is paralyzed, little or no anesthesia and lateral hemianopsia, is probably due to disease in the area supplied by the middle cerebral artery.

(*f*) A lesion causing hemiplegia, hemianesthesia, and lateral hemianopsia is probably situated in the posterior portion of the internal capsule.

(*g*) A lesion causing hemianesthesia, ataxic movements of one-half of the body, no distinct hemiplegia, and lateral hemianopsia could be situated in the posterior lateral part of the optic thalamus.

(*h*) A lesion causing the symptoms of disease of the base of the brain, associated at the same time with changes in the pupil, changes in the nerve-head, and lateral hemianopsia, could be situated in one optic tract or in the primary optic centers on one side.[1]

(*i*) Incomplete hemianopsia, assuming usually a quadrant-shaped defect, may be present on account of a lesion confined to the lower half of the cuneus. It may also occur with less definite limitations in lesions of the subcortical substance of the occipital lobe and then may be associated with other symptoms, as hemiplegia and hemianesthesia. Finally, it may occur from a lesion of the tract, but then will be accompanied by other symptoms indicating basal disease or from a lesion of the external geniculate body.

(*j*) A hemianopsia in which there is preservation of the light-sense, but loss of either the color-sense or the form-sense indicates that the lesion is in the cortex of the visual center.

The Pupil in Hemianopsia.—The reaction of the pupil in hemianopsia has usually been regarded as a localizing symptom of importance; this is denied by some observers (see footnote). Generally the rules are stated as follows:

If, in hemianopsia, the light thrown upon either the blind or the seeing side of the retina causes contraction of the pupil, the lesion is back of the primary optic centers.

If there is no contraction of the pupil when the ray of light falls upon the blind side of the retina, but there is contraction when it falls upon the seeing side, the lesion is in front of the primary optic centers.

[1] The preceding paragraphs have for the most part been condensed from the rules given by Dr. Seguin for the diagnosis of the seat of lesion in cases of hemianopsia.

In the former instance the lesion is so situated that there is no disturbance of the sensorimotor arc of the pupils; in the latter the lesion interferes with this arc, and the pupillary change receives the name *hemiopic* or *hemianopic pupillary inaction.* It is often called *Wernicke's symptom.*[1]

[1] Henschen (Klin. med. anat. Beiträge zur Pathologie des Gehirns, Th. iii) concludes that the hemiopic pupillary inaction (abbreviated H. R.) is present in tract lesions, even when minute or merely caused by pressure; lesions of the posterior segment of the thalamus and pulvinar—perhaps from pressure on the tract, or by destroying the brachium anterius; lesions of the chiasm (occasionally absent from unknown reasons); and in lesions of the nerve, with unilateral hemianopsia. It is a difficult symptom to demonstrate (Henschen uses a special lamp) and its existence is doubted by some observers. The iris reaction may not be entirely absent when the ray falls on the blind side of the retina, but it is much less marked than the one which follows light stimulus of the opposite side. C. B. Walker (Trans. Section on Ophthalmology, Amer. Med. Assoc. 1914,) has designed a clinical instrument for elucidating the hemiopic pupillary reaction and concludes from his studies that it has no topical diagnostic value. To replace Henschen's phenomenon, Wildbrand has proposed a prism test. It is thus described by Saenger: The patient fixes a white point on a large black plane surface. Suddenly two prisms of equal degree (15°) are brought before both eyes, their apices being turned to the hemianopic defect. If cortical hemianopsia exists, the patient's eyes will move so that the fovea is directed to the object; if the reflex path is interrupted by a lesion of the tractus, there is no movement of the eyes.

CHAPTER XIX

MOVEMENTS OF THE EYEBALLS AND THEIR ANOMALIES

Anatomy and Physiologic Action of the Ocular Muscles.—The movements of the eye are controlled by the action of six muscles, four straight and two oblique, in general terms situated in the orbital region.

1. The *external rectus* arises by two heads, respectively from the outer margin of the optic foramen and the common tendon of the inferior and internal recti, and in part from a process of bone on the lower margin of the sphenoid fissure. Its tendon is inserted into the sclera 7 mm. from the margin of the cornea. It is supplied by the *sixth* or *abducens nerve*. Its pre-eminent[1] muscular action is *abduction*—that is, it rotates the eye directly outward.

2. The *internal rectus* arises from the optic foramen by a tendon common to it and the inferior rectus, and passes forward to be inserted by a tendinous expansion into the sclerotic coat 5 mm. from the margin of the cornea. It is supplied by one of the three branches of the inferior division of the *third* or *oculomotor nerve*. Its pre-eminent muscular action is *adduction*—that is, it rotates the eye directly inward.

3. The *superior rectus* arises from the upper margin of the optic foramen and from the fibrous sheath of the optic nerve, and is inserted by a tendinous expansion into the sclerotic coat 8 mm. from the margin of the cornea. It is supplied by the superior division of the *third* or *oculomotor nerve*. Its pre-eminent muscular action is *elevation* or *superduction*—that is, it rotates the eye upward. It also adducts it and rotates the upper end of the vertical meridian of the cornea inward (*inward torsion* or *intorsion*).

4. The *inferior rectus* arises from the optic foramen by a tendon common to it and the internal rectus and passes forward to be inserted by a tendinous expansion into the sclerotic coat, 6 mm. from the margin of the cornea. It is supplied by one of the three branches of the inferior division of the *third* or *oculomotor nerve*. Its pre-eminent muscular action is *depression*, or *subduction*—that is, it rotates the eye downward. It also adducts it and rotates the vertical meridian of the cornea outward (*outward torsion, extorsion*).

5. The *superior oblique* (trochlear) is situated at the upper and inner side of the orbit, and arises above the inner margin of the optic foramen. It proceeds to the inner angle of the orbit, at which point its rounded tendon passes through a fibrocartilaginous pulley occupying a fossa just within the supra-orbital margin of the frontal bone, and is next

[1] This term is borrowed from Maddox.

reflected backward, outward, and downward, to be inserted about 18 mm. from the edge of the cornea between the superior and external recti. It is supplied by the *fourth* or *trochlear nerve*. Its pre-eminent muscular action is *intorsion*—that is, it rotates the vertical meridian inward. It also depresses the eye and abducts it.

6. The *inferior oblique* is situated at the bottom of the orbit and arises from a depression in the orbital plate of the superior maxillary bone. Passing beneath the inferior rectus, it is directed outward, backward, and upward, and reaches its insertion into the sclera by means of a thin tendon about 19 mm. from the corneal margin, within the position of the external rectus. It is supplied by the largest branch of the superior division of the *third* or *oculomotor nerve*. Its pre-eminent muscular action is *extorsion*—that is, it rotates the vertical meridian outward. It also elevates the eye and abducts it.[1]

The starting-point from which the actions of the muscles are reckoned is the *primary position* of the globe, defined by Mauthner as that position of the eyes from which the visual lines can be moved without the eyes being revolved around their anteroposterior axes. The eyes occupy about this position when they are directed straight forward, the head being held erect, and a distant object, situated in the median line of the visual plane, is observed with practically parallel visual lines. Positions of the eyes other than this are called *secondary positions*.

Rotation of the Eyeball Around the Visual Line.—If a *vertical line* is passed through the *visual line* so as to divide the eyeball into two lateral halves, it will intersect the surface of the eyeball in what is called the *vertical meridian*. The latter may be defined with sufficient accuracy as a line passing through the center of the pupil in a direction perpendicular to the line joining the centers of the two pupils. It joins the uppermost and lowermost points of the corneal margin.

In movements of the eyeball directly upward (combined action of the superior rectus and inferior oblique) or downward (combined action of the inferior rectus and superior oblique), or directly inward or outward, the vertical meridian remains vertical.

In *oblique* movements of the eyeball, upward and inward (superior and internal rectus, with inferior oblique); downward and inward (inferior and internal rectus, with superior oblique); upward and outward (superior and external rectus, with inferior oblique); or downward and outward (inferior and external rectus, with superior oblique), the vertical meridian will be observed to rotate like the spokes of a wheel (*wheel-rotation* or *torsion*). The eyeball appears to rotate around the visual line; this is effected by the superior and inferior recti and the superior and inferior oblique muscles. The upper extremity of the vertical meridian of the cornea is deviated outward (toward the temple) by the inferior recti and inferior oblique muscles; and inward (toward the nose) by the superior recti and superior oblique muscles. The

[1] Duane, basing his opinion on the results of paralysis, believes that *depression* is the most important muscular action of the superior oblique and that *elevation* is the most important action of the inferior oblique, intorsion and extorsion, respectively, being subsidiary actions.

deviation of the vertical meridian is greatest when the axis of rotation coincides with the visual line.

The superior and inferior recti exercise the greatest degree of torsion when the eyeball is drawn toward the nose, and either upward or downward.

The oblique muscles, on the contrary, exercise their maximum amount of torsion when the eyeball is drawn toward the temple, and either upward or downward. The inferior oblique, while it aids the superior rectus in upward movements, antagonizes it in the rotation of the vertical meridian and the movement of the eyeball inward.

The visual line coincides most nearly with the axis of rotation of the superior and inferior recti when the eyeball is drawn toward the nose and most nearly with that of the superior and inferior oblique muscles when the eyeball is turned toward the temple. The superior oblique aids the inferior rectus in drawing the eye downward, but antagonizes it in the rotation of the vertical meridian and in the movement of the eyeball inward.

In extreme diagonal movements of the eyes the action of the obliques and the recti is to make the vertical meridian tilt toward the nose or temple. But if the muscles are evenly balanced, the vertical meridians of the two eyes, however tilted, remain parallel, and under these conditions the retinal images are projected normally, vertical objects still appearing vertical.

In paralysis of the eye muscles the vertical meridians no longer remain parallel, and the image of one eye appears oblique with regard to the other. (See also page 526.)

Associated Movements.—Except in pathologic circumstances, there is coördination in the movements of the eyes, and the movement of one eyeball is associated with that of its fellow. In other words, both eyes are used for seeing (*binocular vision*), and are so adjusted that the image of the object regarded falls simultaneously on both maculas (*binocular fixation*). If a distant object is to be looked at and the right eye is turned to the right, the left eye is also turned to the right and to the same extent as its fellow, because of the associated action of the external rectus of the right eye and the internal rectus of the left eye under the same innervation-impulse. If one eye is elevated, the other is also elevated; if one is depressed, the other is also depressed. These are associated movements in the same direction.

If a near object is to be looked at, the visual axes converge for the point at which it is situated because of the associated action of the internal recti of the two eyes (*convergence* or *accommodative* movement); if the eyes are removed from this point and directed to a distant object, the visual axes tend to parallelism because of the action of both external recti.

If the associated movements of the eyes were not thus regulated by equal impulses from the coördinating center, single vision would not be possible, because the images of any object would not fall upon *corresponding points* of the two retinas. Inasmuch as every normal individual has two normally constructed eyes, he must receive from every object

two sets of sensations, which are blended into one when the movements of the eyes are so arranged that the images fall upon corresponding retinal areas.

A point situated anywhere upon the right side of one retina has its corresponding point upon the right side of the other retina, and points on the left side of one correspond with points on the left side of the other. The upper half of the retina of the right eye corresponds to the upper half of the retina of the left eye, and the lower half of the right to the lower half of the left; the nasal side of the right eye corresponds with the malar side of the left, and the malar of the right with the nasal of the left. If, for any reason, the movements of the eyes become disarranged so that the images do not fall upon corresponding or identical retinal areas, the images become double.

The desire for *binocular single vision*, or single vision with the two eyes, which depends upon the blending of the two sets of sensations, or, as it is also called, *fusion*, is believed to be the origin of the impulse which directs the movements of the eyeballs, especially in association in the same direction.

In addition to this desire for blending the two sets of sensations into one, seen in the associated movements of the eyes in the same direction, there is also another regulating factor—*i. e.*, the connection between convergence and accommodation (see page 43).

Overcoming Prisms.—When a prism is placed before one eye with its base inward and diplopia is produced, an outward rotation of the eye occurs, and when the prism is placed with its base outward, an inward rotation of the eye takes place, and the influence of the prism is overcome, so that single vision again is possible within the limitations which have been recorded on page 74, where *prism-convergence*, *prism-divergence*, and *sursumvergence* are described.

Field of Fixation.—This includes all points which the eye under observation can successively fix, the head being perfectly stationary. The field of fixation of an amblyopic eye may be determined by watching the image of a candle-flame on the center of the cornea as the eye follows the test-light moved along the perimeter arc until the limit of movement is reached. Ordinarily the patient should be seated in the position for testing the visual field before the perimeter, with the semicircle horizontal, and the eye (the head being rigid) made to follow a word composed of small test-letters, and the point where vision ceases to be distinct marked on successive meridians. In place of letters, two fine dots set close together on a card may be employed, and the point noted where the dots cease to appear as two.

Landolt's measurements of the field of fixation under normal conditions are as follows: Outward, 45–50; inward, 45; upward, 35–40; downward, 60. Duane's average measurements are: Outward, 51; inward, 53; upward, 43; downward, 63.

G. T. Stevens determines the rotations of the eyes with a special instrument called a *tropometer*. According to him, the most favorable rotations are: Outward, 50; inward, 50; upward, 33; downward, 50.

Strabismus, Squint, or Heterotropia.—Under the general term *strabismus* or *squint* are included those conditions which occur when the visual axis of one eye is deviated from the point of fixation. The eye the visual axis of which is directed to the object fixed is termed the *fixing eye;* the other eye is termed the *squinting* or *deviating eye.* The deviation may be inward (*strabismus convergens*), outward (*strabismus divergens*), upward (*strabismus sursum vergens*), or downward (*strabismus deorsum vergens*).

1. **Convergent Strabismus, or Esotropia.**—In this form of squint the visual line of one eye is directed to the object fixed. The visual line

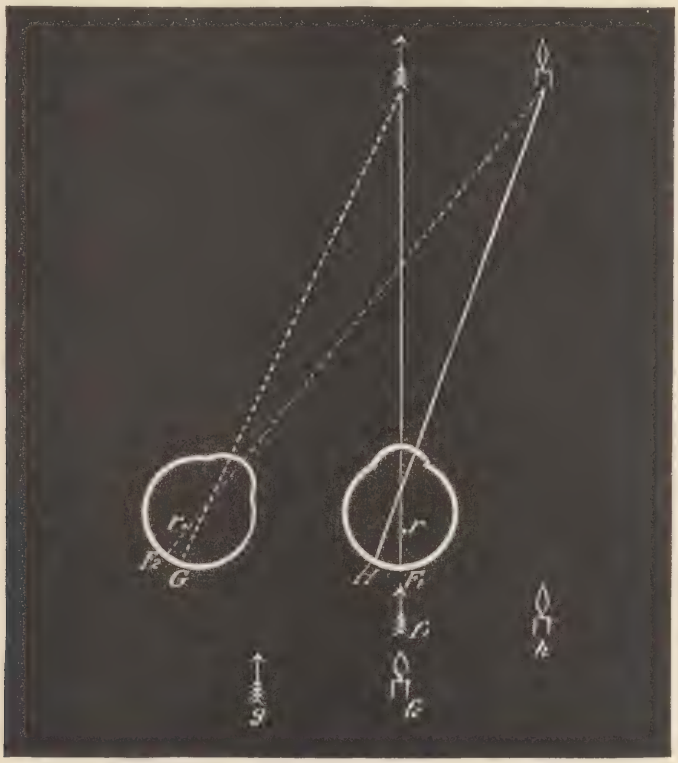


Fig. 229.—Convergent strabismus. Position and projection of the images (James Wallace).

of the other eye is deviated inward, and intersects that of the sound eye at some point nearer than the object fixed. The image of an object situated on the visual line of this eye would be formed on the fovea, and projected to the same point in the field of fixation.

Figure 229 represents a convergent squint of the left eye, and serves to explain the results of an inward deviation of one eye from any cause.

The center of rotation is seen at *r*. The arrow is the object fixed; its image is formed on the fovea of the right eye, F_1, and its position in the field is denoted by f_1. The candle forms its image on the retina of the right eye to the left of the fovea at *H*;

its image is properly projected to the right, and its position in the field is denoted by *h*. The visual axis of the left eye is directed to the candle; its image is formed on the fovea at F_2, and its position in the field is denoted by f_2, identical with that of f_1, because formed on an identical point of the retina. The arrow forms an image on the retina of the left eye at *G*, to the right of the fovea; it is consequently projected to the left of that of F_2, and its position in the field is denoted by *g*.

The right eye projects the images correctly; the left eye projects them to the left of their true position—*i. e.*, to the side of the squinting eye. The diplopia is *simple* or *homonymous*.

2. **Divergent Strabismus, or Exotropia.**—In this form of squint the visual line of one eye fixes the object, while the visual line of the

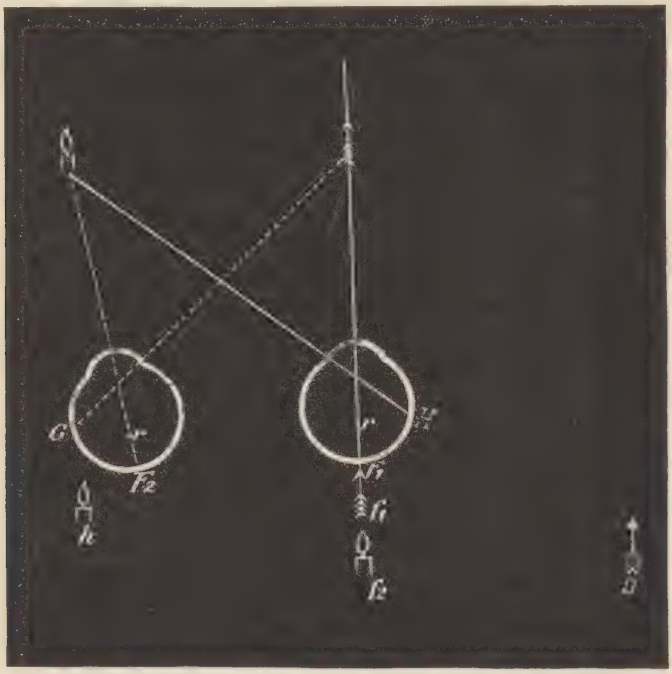


Fig. 230.—Divergent strabismus. Position and projection of the images (James Wallace).

other eye lacks the necessary movement inward to intersect that of its fellow at the point of fixation.

As long as the visual axis of the affected eye intersects that of the sound eye in its anterior extremity, the affection may be denominated *insufficiency of convergence*. When the visual axes no longer intersect anteriorly, but diverge from each other so that their posterior extremities intersect, the affection may be denominated *divergent squint*.

Figure 230 represents a divergent squint of the left eye, and explains the effects of an outward deviation of one eye from any cause upon the position of the images of an object which is fixed.

The center of rotation is at r. The arrow is the object fixed; its image is formed on the fovea of the right eye at F_1, and its position in the field is denoted by f_1. The candle forms its image on the retina of the right eye to the right of the fovea at H; its image is properly projected to the left and its position in the field is denoted by h. The visual axis of the left eye is directed to the candle; its image is formed on the fovea at F_2, and its position in the field denoted by f_2, identical with that of f_1, because formed on identical points of the retina. The arrow forms an image on the retina of the left eye at G, to the left of the fovea; it is consequently projected to the right of that of F_2, and its position in the field is denoted by g.

The right eye projects the images properly; the left eye projects them to the right of their true position—*i. e.*, the side opposite to the squinting eye. The diplopia is *crossed* or *heteronymous*.

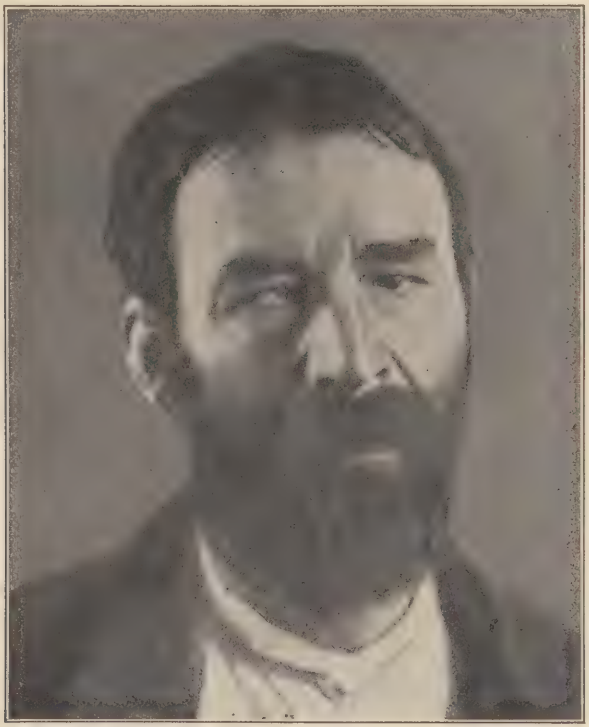


Fig. 231.—Convergent strabismus with decided upward deviation (from a patient in the Philadelphia General Hospital).

3. **Upward and Downward Squint, or Hypertropia.**—If vertical deviation (upward or downward) causes diplopia, the images are on different levels, the upper image corresponding with the lower eye. Simple vertical deviation without lateral is rare. Generally in lateral strabismus the squinting eye deviates upward, but may also turn downward (Schweigger). According to Hansell, functional internal squint (*esotropia*) is always associated with upward deviation (*hyper-*

tropia). In deviations, especially when there is vertical squint, one of the images is often oblique with regard to the other.

This obliquity can be simplified for study by dividing it into two kinds: either the vertical meridians incline toward each other by their upper extremities, or else they diverge from each other.

The meridians *diverge* from each other when the upper extremity of one vertical meridian is directed toward the temple, while the vertical meridian of the other eye remains perpendicular. An object extending in a vertical direction—for example, a candle—would form an inverted image on the retina of the eye whose vertical meridian is tilted toward the temple by its upper extremity, in which the flame of the candle would occupy the lowest portion of the image lying somewhat on the temporal half of the retina, while the lower portion of the candle would occupy the highest portion of the image, somewhere on the nasal half of the retina. In accordance with the law of projection, images on the nasal half of the retina are referred to the temporal portion of the field, and images on the temporal half of the retina are referred to the nasal portion of the field. With the vertical meridian tilted toward the temple the candle forms an image on the retina which is projected outward, so that it seems to converge by its upper extremity toward that of the other eye when the diplopia is homonymous; when crossed diplopia exists, it seems to diverge.

The meridians *converge* toward each other when the upper extremity of one vertical meridian is tilted toward the nose, while the vertical meridian of the other eye remains perpendicular.

When the vertical meridian is tilted toward the nose by its upper extremity, the image of the candle occupies, with its lower portion, a point in the nasal half of the retina, and with its upper portion a point in the temporal half of the retina. It is projected outward in such a manner that it seems to lean away from the image of the other eye when the diplopia is homonymous; when crossed diplopia exists, it seems to lean toward the image of the other eye.

In paralysis of the ocular muscles it is usually the image of the paralytic eye which appears oblique. Sometimes, however, the patient regards this image as vertical and the image of the sound eye as oblique (see also pages 520 and 521).

Paralysis of the Exterior Ocular Muscles (*Paralytic Strabismus*). —This may be *complete* (the muscle is entirely paralyzed) or *incomplete* (the muscle is partially paralyzed).

A. **General Symptoms.**—Certain symptoms are common to paralysis of the external eye muscles.

1. *Loss of Binocular Single Vision, or Diplopia.*—The cause of this, evident from the previous explanations, depends upon the deviation of the affected eye so that the images from an object are no longer fused, owing to their failure to fall upon "identical points" in the two retinas. Diplopia increases as the object is moved to the side of the paralyzed muscle. In slight degree it amounts only to indistinct vision.

2. *Non-correspondence of the Direction of the Two Eyes, or Strabismus.*

—This depends upon the deviation to which the affected eye is subjected by the tone of the unresisted action of the muscle which is the antagonist of the paralyzed muscle, and also, in part, upon the effect of secondary contractures. Squint is not always plainly manifest, and may appear only if an attempt is made to move the eye in the direction of the action of the palsied muscle.

3. *Loss or Limitation of Movement* (*Primary Deviation*).—The limitation of movement is always in the direction of the action of the affected muscle; consequently the deviation of the eye is in a direction opposite to the action of the muscle.

4. *Deviation of the Sound Eye, While the Affected Eye Fixes* (*Secondary Deviation*).—During the act of fixation by the affected eye the same degree of nervous impulse passes from the center to the muscles of the affected eye and to those of its non-affected associate; the former requires an abnormally great impulse to stimulate its movement, and hence the latter is overexcited, and the resulting movement is excessive. The secondary deviation, therefore, is greater than the primary deviation.

In order to demonstrate this the sound eye is covered with the hand, while the affected eye is directed toward an object held at a distance of about one foot. The covering hand is then moved from the sound to the affected eye. In order to fix the object, the sound eye must now move in a direction opposite to that toward which the paralyzed muscle rotates the ball. This backward movement represents the degree of previous excess called into existence by the undue amount of nerve-force which the normal muscle originally received. Thus primary and secondary deviations are in opposite directions, but both in the line of action of the affected muscle.

5. *False Projection of the Field of Vision.*—This depends upon an inaccurate estimation of the position of an object situated in such a portion of the visual field that it requires an effort on the part of the affected muscle to turn the eye toward it. A normal individual (his head being stationary, and one eye being closed, *e. g.*, the right) can readily and accurately touch an object lying within his reach to the left of the median line, because the degree of innervation required to make the lateral movement of the eye in order to see the object gives the necessary information, based on experience, how far to the left the object lies. In the same circumstances an individual with a paretic left external rectus, instead of touching the object, would pass his hand beyond it—*i. e.*, to the left of it, because the excessive innervation which is now necessary to make the lateral turn gives the impression that the object lies farther to the left. In other words, the object is projected to a position in the visual field which it does not have.

6. *Vertigo.*—This depends, both eyes being open, upon the diplopia and the confusion arising from trying to distinguish between the real and the false image. If one eye (the unaffected eye) is closed, it depends upon the condition described in the preceding paragraph.

In a paretic condition of the muscles which rotate the eye down-

ward vertigo may result from an erroneous localization of objects in the lower field, as they seem to lie in a plane deeper than they really are. For these reasons patients with ocular palsies commonly close the affected eye, although closure of either eye would remove the diplopia.

7. *Altered Position of the Carriage of the Head.*—This depends upon the impulse of the patient to carry his head in that direction in which he is least troubled by the double images, and this is usually in the direction toward which the affected muscle moves the eye. In vertical deviation the head is often tilted toward one shoulder—toward the side of the higher eye if the hyperphoria or hypertropia is combined with crossed diplopia, and toward the other side if the hyperphoria or hypertropia is combined with homonymous diplopia.

B. **Varieties of Diplopia.**—There are two varieties of diplopia, according to the relation which the double images bear to the eyes. If the right image pertains to the right eye, and the left image to the left eye, the diplopia is designated *simple* or *homonymous;* if the right image pertains to the left eye, and the left image to the right eye, the diplopia is named *crossed* or *heteronymous.* The explanation of these conditions has been given (see Figs. 229 and 230).

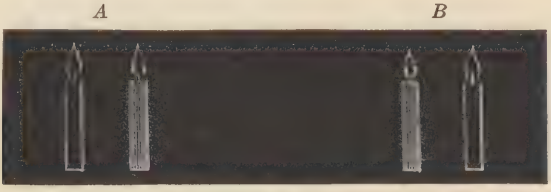


Fig. 232.—*A*, Position of images in paralysis of left external rectus, and *B*, in paralysis of right externus. The false image is drawn in outline (after Fuchs).

If the two images are on a horizontal line, the phenomenon is called *horizontal diplopia.* Vertical displacement of the double images constitutes *vertical diplopia* (see also page 526).

C. **Special Symptoms.**—The following paragraphs contain the most important symptoms peculiar to paralysis of individual muscles. For convenience it is supposed that the *right* eye is affected.

1. **External Rectus.**—The following phenomena may be present:

(*a*) *Horizontal homonymous diplopia,* the images being side by side and parallel, if the eyes are directed on a horizontal level, the distance between them widening as the test-object is moved to the right—that is, the maximum diplopia is to the right (Fig. 232).

If the test-object is moved to the right and above, and the eyes are directed toward it, the false image (image of the right or affected eye) diverges from the real image (image of the left or unaffected eye). This occurs because, in these circumstances, the movement of the right eyeball toward the temple is limited by the feeble external rectus, and the eyeball fails to come into the position where the inferior oblique has its favorable condition for rotating the vertical meridian outward; hence the vertical meridian remains near to a perpendicular, while that

of the sound eye is tilted toward it. There is divergence of the vertical meridians (the false image converges toward the real one) when the eyes are directed downward and toward the right, because the eyeball fails to come into a favorable position to have its vertical meridian tilted toward the nose by the superior oblique, while that of the other eye is tilted toward the temple by the inferior rectus.

(*b*) *Convergent strabismus*, which increases as the eye attempts to follow an object which is moved toward the right, during which it will be noticed that there is *limitation of movement* in this direction.

(*c*) The *secondary deviation* of the sound eye is inward; the *false projection of the field of vision* is to the right side, and the *face is turned* to the *right—i. e.*, to the side of the affected muscle.

2. **Internal Rectus.**—There are present:

(*a*) *Horizontal crossed diplopia*, the images being side by side and parallel, if the eyes are directed along a horizontal level, the distance between them widening as the test-object is moved to the left, or if the eyes are directed upward—that is, the maximum diplopia is to the left (Fig. 233).

If the test-object is moved to the left and above, and the eyes are directed toward it, the image of the affected eye is lower than that of the

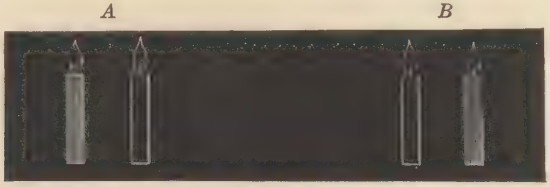


Fig. 233.—*A*, Position of images in paralysis of left internal rectus, and *B*, in paralysis of right internus. The false image is drawn in outline (after Fuchs).

unaffected eye, and its upper extremity inclines toward it; if the test-object is moved to the left and downward, the false image is higher and its lower extremity inclines away from that of the real image. These inclinations occur because, in these circumstances, the left eyeball is placed in a favorable position for one of the oblique muscles to rotate it, while the right eye is not brought in sufficiently for the superior or inferior rectus to exercise its torsion effect; consequently, the vertical meridians diverge on looking upward and converge on looking downward toward the left side.

(*b*) *Divergent strabismus*, which increases when the eye attempts to follow an object moved to the left, during which it will be noticed that there is *limitation of movement* in this direction.

(*c*) The *secondary deviation* of the sound eye is outward, the *false projection of the visual field* is to the left side, and the *face is turned* to the *left—i. e.*, to the side of the affected muscle.

3. **Superior Rectus.**—There are present:

(*a*) *Vertical crossed diplopia* in the upper field, the images being one above the other, the image of the affected eye being higher than its

fellow and inclined to the left (healthy side), and the vertical distance between them (difference in height) widening as the test-object is moved upward and to the right—that is, there is maximum diplopia in looking up and to the right (Fig. 234).

If the test-object is moved upward, and to the left, and the eyes are directed toward it, the obliquity of the images increases—*i. e.*, the false image is still more inclined toward the sound side, away from that of the other. This occurs because, in these circumstances, the inferior

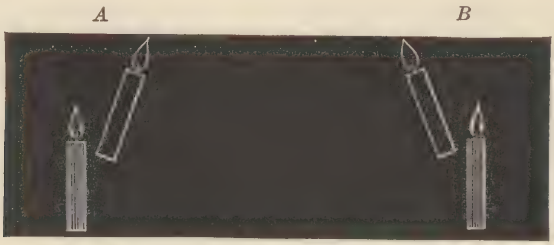


Fig. 234.—*A*, Position of images in paralysis of left superior rectus, and *B*, in paralysis of right superior rectus (Fuchs).

oblique rotates the vertical meridian of the sound eye to the left, while the affected eye, owing to the loss of power in the superior rectus, is unable to deviate its vertical meridian from the perpendicular; therefore the two meridians diverge, but, the diplopia being crossed, the images also diverge.

(*b*) *Downward strabismus*, which increases when the eye attempts to follow an object moved upward, during which it will be noticed that there is *limitation of movement* in this direction.

Fig. 235.—*A*, Position of images in paralysis of left inferior oblique, and *B*, in paralysis of right inferior oblique (after Fuchs).

(*c*) The *secondary deviation* of the sound eye is upward, the *false projection of the visual field* is too high, and the *face is directed upward and to the right*, or the *head is tilted toward one shoulder*, generally the left.

4. **Inferior Oblique.**—There are present:

(*a*) *Vertical homonymous diplopia* (*sometimes crossed*) in the upper field, the images being one above the other, the image of the affected eye being higher than its fellow and inclined to the right—*i. e.*, to the affected side—the vertical distance between them (difference in height)

widening as the test-object is moved upward and to the left—that is, there is maximum diplopia on looking up and to the left.

If the test-object is moved upward and to the right and the eyes are directed toward it, the obliquity of the images increases—*i. e.*, the false image is still more inclined away from the sound side. This occurs because, in these circumstances, the vertical meridian of the right eye is not tilted toward the temple, owing to loss of power in the inferior oblique, while that of the left eye is tilted toward the nose by the superior rectus, now in its best position for tilting the vertical meridian inward; therefore the two meridians incline toward each other by their upper extremities.

(*b*) The *direction of the affected eye* is downward and inward, which is more noticeable when the eye attempts to follow an object moved upward and outward, during which it will be noticed that there is *limitation of movement* in this direction.

(*c*) The *secondary deviation* of the sound eye is upward and inward, the *false projection of the visual field* is too far upward, and the *face is directed upward* and *toward the left,* or the *head is tilted* toward one shoulder.

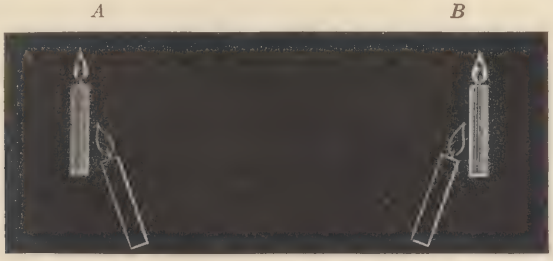


Fig. 236.—*A*, Position of images in paralysis of left inferior rectus, and *B*, in paralysis of right inferior rectus (after Fuchs).

5. **Inferior Rectus.**—There are present:

(*a*) *Vertical crossed diplopia* in the lower field, the images being one above the other, the image of the affected eye being lower than its fellow and inclined to the right—*i. e.*, to the affected side—and the vertical distance between them (difference in height) widening as the test-object is moved downward and to the right—that is, there is maximum diplopia on looking down and to the right.

If the test-object is moved downward and to the left, and the eyes are directed toward it, the obliquity of the images increases—*i. e.*, the false image inclines still more toward the affected side. This occurs because, in these circumstances, the superior oblique of the left eye is in its best position for rotating the vertical meridian toward the nose; but the right eye, by reason of its paralyzed inferior rectus, is unable to tilt its vertical meridian to correspond; therefore the vertical meridian of the right eye remains perpendicular, while that of the left eye inclines toward it. The image of the right eye seems to be the oblique one; the images diverge, but, the diplopia being crossed, they seem to converge.

(*b*) *Upward strabismus*, which increases when the eye attempts to follow an object moved downward, during which it will be noticed that there is *limitation of movement* in this direction.

(*c*) The *secondary deviation* of the sound eye is downward and outward, the *false projection of the visual field* is too far downward, and the *face is directed downward* and to the right, or the *head is tilted* toward one shoulder, generally the right.

6. **Superior Oblique.**—There are present:

(*a*) *Vertical homonymous diplopia* (*sometimes crossed*) in the lower field, the images being one above the other, the image of the affected eye being lower than its fellow, and inclined to the left—*i. e.*, to the sound side—the vertical distance between them (difference in height) widening as the test-object is moved downward and to the left—that is, there is maximum diplopia downward and to the left.

If the test-object is moved downward and to the right, and the eyes are directed toward it, the obliquity of the images increases—*i. e.*, the false image inclines still more toward the sound side. This occurs because, in these circumstances, the vertical meridian of the left eye

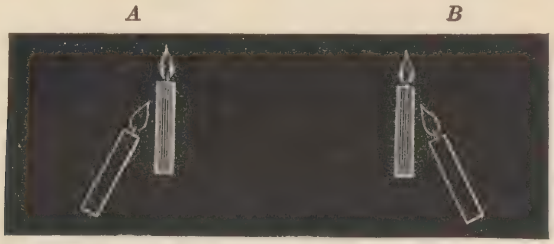


Fig. 237.—*A*, Position of images in paralysis of left superior oblique, and *B*, in paralysis of right superior oblique (after Fuchs).

is inclined toward the left by the inferior rectus, while that of the right eye is not rotated, owing to the feeble superior oblique; consequently, the meridians diverge.[1]

(*b*) The *direction of the affected eye is upward and inward*, and is more noticeable when the eye attempts to follow an object moved downward and outward, during which it will be noticed that there is *limitation of movement* in this direction.

(*c*) The *secondary deviation* of the sound eye is downward and inward, the *false projection of the visual field* is too far downward, and the *face is inclined downward* and to the *left*, or the *head is tilted* toward one shoulder, generally the left.

7. **Oculomotor Paralysis.**—There are present:

(*a*) *Complete crossed diplopia*, the image of the affected eye being higher than its fellow, and its upper extremity inclined to the right—

[1] In paralysis of the inferior rectus the diplopia is usually crossed; this feature helps to distinguish it from paralysis of the superior oblique. In both, the image of the affected eye sometimes seems to stand nearer to the patient than the other image. It should be remembered, however, as Maddox insists, that in paralysis of any one of the obliques a pre-existing exophoria may complicate the case to such an extent as to change "homonymous" into "crossed" diplopia, while in paralysis of the superior and inferior recti pre-existing esophoria may convert "crossed" into "homonymous" diplopia.

i. e., to the affected side—the distance between them—*i. e.*, the lateral distance—widening as the test-object is moved to the left. If the test-object is moved upward the difference in height and the inclination of the false image increase.

(*b*) *Divergent strabismus* and *limitation of movement* in all directions, except outward and slightly downward.

(*c*) The *secondary deviation* of the sound eye is outward, the *false projection of the field of vision* is to the inner side, and the *face is inclined* toward the *right*, the chin being tipped *upward*. In addition, there are ptosis, medium dilatation of the pupil which fails to contract to light, and paralysis of accommodation.

Method of Examination and Diagnosis of the Affected Eye.—If the paralysis is complete, there is little difficulty in making a diagnosis by attention to the prominent symptoms which have been detailed. In the condition is one of partial paralysis (paresis), the diagnosis must be based upon an investigation of the double images.

Fig. 238.—Double oculomotor palsy (from a patient in the Philadelphia General Hospital).

The patient is seated with the head and eyes in the primary position, four meters from the test-object (a candle flame or small electric light), and a trial-frame one side of which carries a red glass is placed in position. Hence if diplopia is developed, one image will be yellow and the other red. The lighted candle is then moved from the median line to the right, to the left, upward and down, while the patient follows these movements with his eyes, the head being stationary. By these maneuvers the following facts will be ascertained:

(1) Double images are chiefly seen when the eyes are turned in a direction requiring an action of the affected muscle. (2) The image of the affected eye (false image) is projected in a direction toward which the paralyzed muscle normally rotates the eye. (3) That image is false (image of the affected eye) which travels farther away from the true image (image of the sound eye) when the test-object is moved in the direction of the paralyzed muscle—*i. e.*, the relative distance of the double images increases in these circumstances.

The effect upon the obliquity of the images and their relation to each other of moving the test-object in oblique directions above and below the horizontal plane must next be studied; also whether the images are present in all portions of the field of fixation, or confined to a certain area of it (see also page 526).

Many tables have been prepared to aid in the diagnosis of the affected muscle, and if paralysis of the oblique muscles always produced homonymous or simple diplopia, and paralysis of the superior and inferior rectus muscles always caused heteronymous or crossed diplopia, their construction would be comparatively simple. This, however, is not the case, and it is well known, as has already been pointed out, that the diplopia from paresis of the obliques may be crossed, and that from paresis of the superior and inferior recti homonymous. Hence Duane insists that paralysis of the obliques and of the superior and inferior recti should be diagnosticated from the behavior of the vertical diplopia.

This author divides the twelve muscles moving the two eyes into *three groups* of four each: four moving the eyes laterally, four moving them up (elevators), and four moving them down. Each group is divided into *two pairs*. Thus, the four *laterally acting muscles* are divided into (*a*) a pair of *right turners* (right externus and left internus), and (*b*) a pair of *left turners* (right internus and left externus). The four *elevators* are divided into (*a*) a pair of *right-hand elevators* (right superior rectus and left inferior oblique) and (*b*) a pair of *left-hand elevators* (right inferior oblique and a left superior rectus). The four *depressors* are divided into (*a*) a pair of *right-hand depressors* (right inferior rectus and left superior oblique) and (*b*) a pair of *left-hand depressors* (right superior oblique and left inferior rectus).

In order to assist in the diagnosis of the affected muscle the following table has been constructed by Dr. Duane which the author has found to be most satisfactory, and which Dr. Duane permits him to insert:

TABLE OF DIPLOPIA IN OCULAR MUSCLE PARALYSIS, ACCORDING TO DUANE

A. There is a lateral (*i. e.*, a homonymous or crossed) diplopia which increases markedly as eyes are carried laterally (to right or left). A laterally acting muscle is paralyzed.
   (*a*) Diplopia increases in looking to the right (= paralysis of a right turner).
      Diplopia homonymous: Paralysis of right externus.
      Diplopia crossed: Paralysis of left internus.
   (*b*) Diplopia increases in looking to the left (= paralysis of a left turner).
      Diplopia crossed: Paralysis of right internus.
      Diplopia homonymous: Paralysis of left externus.

B. There is a vertical diplopia which increases in looking up. An elevator is paralyzed.
   (*a*) Vertical diplopia increases in looking up and to the right (= paralysis of a right-hand elevator).
      Diplopia left (*i. e.*, image of right eye above): Paralysis of right superior rectus.
      Diplopia right (*i. e.*, image of left eye above): Paralysis of left inferior oblique.

(*b*) Vertical diplopia increases in looking up and to the left (= paralysis of a left-hand elevator).

Diplopia left (*i. e.*, image of right eye above): Paralysis of right inferior oblique.

Diplopia right (*i. e.*, image of left eye above): Paralysis of left superior rectus.

C. There is a vertical diplopia which increases in looking down. A depressor is paralyzed.

(*a*) Vertical diplopia increases in looking down and to the right (= paralysis of a right-hand depressor).

Diplopia right (*i. e.*, image of right eye below): Paralysis of right inferior rectus.

Diplopia left (*i. e.*, image of left eye below): Paralysis of left superior oblique.

(*b*) Vertical diplopia increases in looking down and to the left (= paralysis of a left-hand depressor).

Diplopia right (*i. e.*, image of right eye below): Paralysis of right superior oblique.

Diplopia left (*i. e.*, image of left eye below): Paralysis of left inferior rectus.

To illustrate the practical working of the table the following example is quoted: The patient with a red glass before the right eye is directed to observe a candle which is moved in all directions in his field of fixation. If the patient has single vision when he looks down, but has vertical diplopia when he looks up, paralysis of an elevator is inferred. The vertical diplopia increases greatly when he looks up and to the right, and diminishes to almost nothing when he looks up and to the left. The paralysis must affect a right-hand elevator (right superior rectus or left inferior oblique). The red image is higher (left diplopia = right eye below). The paralysis must affect the right superior rectus. If it had been the left inferior oblique, the red image would have been the lower; and if it had been either the right inferior oblique or the left superior rectus, the vertical diplopia would have increased not when the patient looked up and to the right, but when he looked up and to the left.

Causes.—The lesion which causes paralysis of an ocular muscle may have an *intracranial*, *orbital*, or *peripheral* situation. If intracranial, it may be *cerebral*—that is, *cortical*, *nuclear*, or *fascicular* in situation, or else *basal*.

Among the conditions residing in the orbit which produce paralysis of the exterior ocular muscles, the so-called *orbital palsies* are cellulitis, tenonitis, periostitis, tumors, metastatic carcinomatous nodules (Elschnig), hemorrhage, fracture, and affections of the sinuses.

Syphilis causes about one-half of the cases of exterior ocular muscle palsies—according to Alexander, 59.4 per cent. The resulting paralysis may be due to an inflammation or gummatous change affecting the nerves at the base of the brain or in the orbit, or it may be central in origin from disease of the nuclei of the nerves or of the brain in their immediate vicinity; or from lesions in the third ventricle, the aqueduct of Sylvius, or the fourth ventricle. Syphilitic paralysis is generally one of the later manifestations, but it has been noted as early as the sixth month after the primary infection, particularly in the form of ptosis. In rare instances paralysis of the ocular muscles results from inherited syphilis (Graefe, Nettleship, Lawford).

Other causes, some of which at times occasion *central*—that is, *nuclear*—lesions, and at other times act *peripherally*, are rheumatism, gout, diabetes, whooping-cough, influenza, herpes zoster, and certain toxic

agents—for example, lead, alcohol, tobacco, gelsemium, conium, chloral, carbonic acid, and fish- and meat-poisoning (ptomain-poisoning, toxalbumins, botulism, and allantiasis).

The external rectus is the muscle most frequently affected by rheumatism and diabetes and often by influenza. So-called rheumatic palsies, as Mauthner has pointed out, may be followed years after by tabes of the cord, disseminated sclerosis, or paralytic dementia. Although diphtheria usually affects the ciliary muscle, it may attack one or more of the exterior muscles, generally the external rectus. The condition may be bilateral. Rarely complete ophthalmoplegia occurs.

The ocular manifestations of fish- and meat-poisoning are usually paresis or paralysis of accommodation and paralytic ptosis. The exterior muscles may be affected. The lesions are probably usually nuclear.

The diseases and lesions which attack the nerves at the base of the brain, and thus occasion the so-called *basal palsies*, are hemorrhage, meningitis, both simple and tubercular, particularly the latter, abscess, for example, in connection with middle-ear disease, aneurysm, diseases of the cavernous sinus, pituitary body disease, syphilis, and tumors. *Spinal anesthesia* may be followed by palsy of the exterior ocular muscles, the external rectus being most frequently involved. It probably depends upon a toxic reaction in the meninges.

A number of paralyses of the exterior ocular muscles are seen in connection with locomotor ataxia, paretic dementia, disseminated sclerosis, and bulbar paralysis. Tabetic paralysis is often transitory in its nature; it may be associated with the pupillary changes characteristic of this affection. Relapses are frequent. Sherrington has demonstrated that the third, fourth, and sixth nerves contain afferent fibers which are not derived from the fifth nerve, and concludes that tabetic ocular palsies are probably due to implication of these afferent fibers and not to the implication of the efferent fibers of the ocular nerves. Paralyses of the orbital muscles of *cerebral* origin may result from degenerative, hemorrhagic, or neoplastic lesions affecting the cortex of the brain, the corticopeduncular region, the nuclei of the nerves, or the nuclear fibers.

Injuries may cause ocular muscle palsy—for example, the muscle may be torn or the nerve-trunk divided, or there may be paralysis owing to periostitis of the orbit, fracture of the orbital walls or base of the skull. The palsy may develop secondarily from basal meningitis, abscess, or nuclear degeneration.

Anomalies of the external muscles occur, depending upon their abnormal insertion. Entire absence of a muscle has been noted. Occasionally cases of orbital muscle palsy have been attributed to various so-called *reflex disturbances*.

Recurrent Oculomotor Paralysis (*Ophthalmoplegic Migraine*—Charcot).—The symptoms of this comparatively rare affection are violent unilateral headache, nausea, vomiting, slight fever, and usually paralysis of the third nerve on the same side as the pain. The attacks

come in periodic crises, and the disease may last from several days to long periods of time. Occasionally the paralysis remains permanent. The lesion is probably one involving the root of the third nerve. Recurrent paralysis of the abducens has been observed.

Congenital Paralysis (*Congenital Deviation*—Duane).—Congenital palsies of the exterior ocular muscles, usually stated to be uncommon, are comparatively frequent if slight deviations are taken into account. They have been particularly well studied by Duane.[1] According to him, insufficiency of the superior rectus, with fixation with the paretic eye and secondary deviation (upshoot) of the other eye, is not infrequent. A striking feature is that when the fixing eye moves outward, the other shoots spasmodically up and in (spasmodic action of the inferior oblique). Other groups described by Duane are palsy of the superior rectus with fixation with the non-paretic eye; slight and marked insufficiency of the inferior rectus; insufficiency of the inferior and superior oblique and combined insufficiencies of the vertical and lateral muscles. Congenital palsy of one or both externi has been described, and may be associated with retraction movements (see next paragraph). The symptoms of congenital palsy usually are: head-tilting, diplopia, vertigo, and asthenopia. The patients nearly always close the defective eye.

Retraction Movement of the Eyeball, Associated with Congenital Deficiency of Abduction.—Certain cases of congenital deficiency of movement of the eyeball are characterized by all or some of the following symptoms, which have been thus summarized by Duane: Usually complete, occasionally partial, absence of outward movement of the affected eye; partial defect of inward movement of the affected eye; retraction of the affected eye into the orbit when it is adducted; a sharp, oblique movement of the affected eye up and in or down and in when it is adducted; partial closure of the eyelids of the affected eye when it is adducted; and paresis or, at least, marked deficiency of convergence, the affected eye remaining fixed in the primary position, while the sound eye is converging.

The affection is undoubtedly congenital. Females are more usually affected than males. The affection has been explained by assuming that the externus is replaced by an elastic or inelastic strand of connective tissue, or that a faulty insertion of the internus causes it to act as a retractor. The oblique upward and downward movements of the eye observed in many of these cases during adduction is attributed by Duane to a spasmodic action of one of the obliques. Operative procedures have been suggested for cosmetic purposes—namely, tenotomy of the retracted muscle and fixation of the globe in the opposite position (Wolff).

It is often difficult to ascertain whether the paralysis is *central* or *peripheral* in its origin. The differential diagnosis must be made by examining into the completeness of the paralysis and the existence of complications or associated symptoms. Peripheral palsies are more apt

[1] Transactions of the American Ophthalmological Society, Vol. XII, Part iii, 1911.

to be isolated and complete; those of central origin are often associated with other symptoms indicative of intracranial mischief.

Relative Frequency of Paralysis of the Orbital Muscles.—Paralysis of the abducens (external rectus) is met with most frequently, the next in order of frequency being unilateral paralysis of the oculomotor. After these come paralysis of the superior oblique, inferior rectus, superior rectus, internal rectus, and inferior oblique. However, statisticians differ exceedingly on these points—*e. g.*, Duane ranks the superior rectus next to the external rectus.

The **prognosis** depends upon the cause of the palsy. Some cases of peripheral paralysis, especially those depending upon syphilis and rheumatism, are readily amenable to treatment; in others not only is the paralysis incurable, but the lesion which creates it may be a fatal one. Hence the importance of trying to ascertain the character and situation of the lesion which produces the palsy.

Treatment.—In syphilis the usual remedies are applicable, and in many instances good results follow very large doses of iodid of potassium. Massive doses are often tolerated, and even if the paralysis has existed for a long time, cure may ultimately result. Salvarsan, in the author's experience, is of little or no service in luetic palsy of the exterior ocular muscles, that is, in cases of long standing. In rheumatism, in addition to iodid of potassium, salicylic acid is useful, especially in the earlier stages. It may be given not in combination with, but at the same time as, the iodid. The various causes which have been mentioned furnish the indications for other treatment. Care should always be taken to examine the nasal accessory sinuses for sources of infection.

The great annoyance which is produced by the double images may be remedied by covering the affected eye with a piece of ground glass, which is mounted in a spectacle-frame. If the patient is ametropic, his correcting lens for the opposite eye may be placed in the same frame.

Sometimes prisms may be worn which fuse the double images. The rules for adjusting prisms are given on page 560.

Mechanical treatment has been suggested by Michel, and was tried in this country by Bull. The conjunctiva is seized near the insertion of the affected muscle with forceps, and the eyeball is drawn forcibly, as far as possible, beyond the ordinary limit of contraction, and then back again. The eye is first cocainized. The movements are made daily, and continued for a minute at a time.

Electricity may be tried, the great difficulty being in passing the current through the muscle. Ordinarily, one pole—the cathode—is placed upon the closed lid, while the other is put upon the temple. Usually, a current of more than 3 milliamperes is unbearable. This is especially true if the pole is placed directly upon the sclera, the eye first having been cocainized. Very disagreeable flashes of light will usually take place if a current of more than 1 or $1\frac{1}{2}$ milliamperes is employed. If faradism is tried, a very weak current should be selected.

Finally, after all other means have failed, tenotomy has been resorted to, or else advancement of the paralyzed muscle. In many cases

advancement of the paralyzed muscle and tenotomy of the antagonist are necessary. The best results are obtained in the lateral muscles. In case an injured muscle—that is, one torn from its insertion—should be seen soon after the accident, it would be proper to find the ends of the divided muscle and stitch them together.

Ophthalmoplegia.—Although the term "ophthalmoplegia" might with perfect propriety be used to describe all the ocular muscle palsies, it is generally reserved for that class of paralyses of the orbital muscles due to disease of the nuclei of the third, fourth, and sixth nerves. In certain clinical cases it is not possible to determine whether the lesion is nuclear or in the nerves. Ophthalmoplegia may be divided into *acute ophthalmoplegia* or *acute nuclear palsy*, and into *chronic ophthalmoplegia* or *chronic nuclear palsy*. When it so happens that the intra-ocular muscles alone are affected, the term *interior ophthalmoplegia* is sometimes employed, and when the exterior muscles alone are affected, the term *exterior ophthalmoplegia*. When both sets of muscles are involved, the term *total ophthalmoplegia* is appropriate.

Acute ophthalmoplegia is characterized by a rapid paralysis of all ocular muscles, often associated with fever and convulsions. Many of the cases have proved to be fatal. They occur with hemorrhage in the region of the nuclei, or as an acute hemorrhagic polio-encephalitis, the primary cause being tuberculosis, syphilis, ptomain-toxemia, or poisoning from alcohol or sulphuric acid. Acute ophthalmoplegia may be associated with acute poliomyelitis, with bulbar palsy, or with facial palsy. According to Oppenheim, an acute ophthalmoplegia may be the result of a peripheral neuritis of nerves of the ocular muscles. Certain poisons—for example, nicotin, lead, and carbonic acid—may cause an ophthalmoplegia which is not fatal, or, at least, not necessarily fatal, and the same is true of one type which is seen with certain constitutional diseases—for instance, diabetes, syphilis, and influenza. *Transient bilateral ophthalmoplegia* has been described, the symptoms developing rapidly and disappearing completely after one or two months.

Chronic ophthalmoplegia is characterized by loss of power in one or more eye muscles, which may gradually increase until every muscle is paralyzed. Sometimes the levator escapes; indeed, ptosis may be absent. The disease may be *stationary* or *progressive*. It is not always symmetric; it may be unilateral. Chronic ophthalmoplegia may follow an acute palsy, the lesions of which have started chronic degenerative changes; it may appear as a *congenital* and occasionally *hereditary* affection, usually in the form of bilateral ptosis. Thus, A. A. Bradburne observed ptosis, with almost complete loss of ocular movement, in a family where this affection had been present in five generations. Sometimes epicanthus complicates the ptosis. Ophthalmoplegia is seen in association with locomotor ataxia, paretic dementia, progressive muscular atrophy, chronic bulbar paralysis, and disseminated sclerosis. The underlying constitutional condition may be syphilis and sometimes tuberculosis. The disease is essentially chronic, and may last for years. It is more

common in males than in females, and is more serious in children than in adults.

It is usually stated that if the intra-ocular muscles escape, which is not always the case, there is presumptive evidence that the origin of the trouble is nuclear, but, as Mauthner has pointed out, it is not a characteristic sign, and a partial palsy may be peripheral. Siemerling concludes that nuclear disease may be inferred from exterior ophthalmoplegia, if it is not maintained that nuclear palsy must manifest itself as an exterior ophthalmoplegia. In general terms the lesions are degenerative, inflammatory, or hemorrhagic. According to Siemerling, the pathologic states underlying progressive paralysis of the ocular muscles may reside in nuclear disease, in degeneration of the muscles and of the nerve-trunks, the nuclei being intact, and in interruption of the conducting power of the intramedullary roots, muscles, nerve-trunks, and nuclei being uninvolved.

Treatment.—In many instances this is wholly without result. If syphilis is present, the usual remedies are applicable, especially iodid of potassium in massive doses.

Associated Ocular Paralyses.—Sometimes the eyes cannot make certain movements in which they are usually associated, although the directing power of the muscles may be unimpaired when they exercise their function in a different association. In other words, there is paralysis of movement and not of the muscles supplied by a given nerve. Thus the internal recti may be unable to draw the eyes together in the act of convergence, although they may act normally in helping to move the eyes from side to side; or there may be loss of the synchronous lateral movement of the external rectus of one eye and the internal rectus of the other (*conjugate lateral paralysis*), although convergence is normal; or the upward or downward movements of the eye may be lost. Lesions affecting the centers for combined movements may produce such phenomena. According to Spiller, paralysis of lateral associated movement may be caused by a lesion of the posterior longitudinal bundle or of the sixth nucleus; symmetric disease of the nuclei of the affected nerve explains some cases in which the upward and the downward movement are lost. But probably the lesion is most frequently in or near the corpora quadrigemina. Holmes and Sargent have described disturbance of ocular movement with injury of the superior longitudinal sinus; in one group temporary palsy of associated conjugate movements occurred, attributed to implication of the posterior part of each frontal convolution. Typical spasmodic conjugate deviation may be caused by hysteria, and this neurosis may also originate palsy of associated parallel movements.

In apoplexy, if the head is drawn from the paralyzed side and the eyes are also turned to the sound side, the condition is called *conjugate deviation of the head and eyes*. The rule is, according to Prevost, that in lesions of the hemisphere the eyes are turned toward the lesion and away from the paralyzed side, but in lesions of the mesencephalon they are turned away from the lesion and toward the paralyzed side. Should

there be unilateral convulsions, with the eye turned toward the convulsed side, there is an irritative lesion in the hemisphere, but if the head and eyes are turned away from the convulsed side, there is an irritative lesion in the mesencephalon (Landouzy).

Divergence Paralysis.—This condition manifests itself by homonymous diplopia and convergent strabismus when the eyes are fixed upon a distant point. As the test-object approaches the patient, and especially on lateral fixation, there is diminution of the convergent strabismus and the diplopia, and finally a point may be reached where there are single vision and orthophoria, while within this limit there may be exophoria. Cases of this character have been described as secondary to an abducens paralysis, and also ascribed to spasm of convergence and to paralysis of a supposed divergence center. Berry believes the correct diagnosis of this condition to be *spasm of convergence* and not paresis of divergence. Duane, however, maintains that the only satisfactory explanation of the phenomenon is that it is due to a *paralysis of divergence.* It occurs at all ages, and more often in conjunction with hyperopia than myopia. He suggests that a lesion near the two abducens nuclei would cause this condition.

Convergence Paralysis.—This, as an extreme variety of convergence insufficiency, has been referred to (see page 555). The symptoms are: crossed diplopia, divergent strabismus, which increases as the test-object is made to approach the eye, no increase of diplopia either to the right or left, and normal rotation of each eye outward and inward. It has been observed in various central nervous disorders, and notably in locomotor ataxia and disseminated sclerosis.

Paralysis of the Interior Ocular Muscles.—Under the general term *cycloplegia* are included the cases of paralysis of the ciliary muscle. These may or may not be accompanied with dilatation of the pupil.

If the ciliary muscle is paralyzed, the chief symptom is loss of accommodation, precisely as it occurs after the instillation of a mydriatic. The loss of accommodation may be *complete* or it may be *partial;* that is, one or more diopters of the entire amount which is normal at the patient's time of life may still remain. After the fiftieth year it is difficult to detect cycloplegia.

It occurs from a lesion in the trunk of the oculomotor nerve or in the anterior part of its nucleus (consult also oculomotor palsy and ophthalmoplegia). Unilateral cycloplegia is said to be possible under the influence of disease of the ciliary ganglion. Paralysis of accommodation may be caused by affections of the nervous system, infectious diseases, and by intoxications. A very common cause of double paralysis of the ciliary muscle is diphtheria. Cycloplegia is also occasioned by spinal disease, by diabetes, by disease of the accessory sinuses, by disorders of metabolism, by traumatism (usually these are associated with mydriasis), by prolonged eye-work, by mumps, by tonsillitis, and frequently by acquired syphilis, and is often associated with paralysis of the sphincter of the iris. Inherited syphilis is a rare cause of paralysis of accommodation. Paresis of the ciliary muscle is common after cer-

tain fevers—for example, typhoid fever. Various ptomains, toxins, fish, and meat poisonings may cause both paresis and paralysis of the ciliary muscle. Congenital interior ophthalmoplegia is a rare anomaly (Duane). Toxic and traumatic cycloplegias are usually peripheral; syphilitic and parasyphilitic varieties may be peripheral, basal, and nuclear in origin.

Under the general term *iridoplegia* are included the conditions which occur when there is loss either of the direct or of the associated action of the iris, due to paralysis of its sphincter. The chief symptom is connected with changes in the action of the pupil. The condition may or may not be accompanied with paralysis of the ciliary muscle. The various pupillary changes have been discussed in Chapter II, page 55. Consult also page 539.

Concomitant Strabismus or Squint: Heterotropia.—This form of strabismus is characterized by the power of the squinting eye to follow the movements of the other eye in all directions, the angle of squint always maintaining the same size.

Varieties of Concomitant Strabismus.—The chief deviations of squinting eyes, as already given, are: *convergent strabismus*, or *esotropia; divergent strabismus*, or *exotropia;* and *vertical strabismus*, or *hypertropia.* Concomitant squint may be *periodic* or *constant.* The latter variety is divided into *monocular squint*, that is, in ordinary circumstances the same eye always deviates when the other eye is used for fixation, and *alternating squint*, that is, either eye is used indifferently for fixation. Lateral squint is usually associated with upward deviation. It is probable that at first squint is generally periodic, but with repeated recurrences, as Priestley Smith expresses it, the suppression of the deviating image becomes confirmed, and the squint becomes continuous. The average age for squint to begin is three and four-tenth years, although it is often noticeable during the first year of life. Squints occurring after five years are apt to be alternating, in which case excellent vision exists in each eye. According to Duane, a slight vertical congenital deviation may be the starting-point of a progressive lateral deviation, which is unnoted until the child is five or six years of age.

Causes of Concomitant Strabismus.—The etiology of strabismus has occasioned much discussion, and even at this time is not a settled question. In general terms, the factors which have been considered important in the causation of squints may be summarized as follows:

1. Disturbance of the relation between accommodation and convergence by errors of refraction.

2. Inequality in the vision of the two eyes, or amblyopia of one eye, which removes the natural stimulus of diplopia to exact convergence.

3. Disturbances of innervation and defective development of the fusion faculty.

These causes of squint are somewhat elaborated in the succeeding paragraphs.

1. *Disturbances in the Relations of the Functions of Accommodation and Convergence.*—The relation between these two functions has been

previously described (see page 43). Some latitude of movement is possessed by each function separately; but a limit to the independent exercise of either function exists, beyond which neither function can operate alone. Thus, a hyperopia of 6 D would require an accommodation of 6 D to neutralize it, the visual lines being parallel. This is rarely possible; some meter-angles of convergence will usually accompany the accommodative effort. The point of convergence is then nearer than the point accommodated for, constituting a convergent squint. Hyperopia is, therefore, frequently accompanied by convergent squint.

In contrast to this, a myope of 10 D requires 10 meter-angles of convergence to see at his far point of vision, that is, the point at which he can see with relaxed accommodation. This is not usually possible, because the enormous convergence necessary to see at this point is too severe a strain; consequently, the visual lines intersect at a greater distance than the point for which they are accommodated, and binocular vision is abandoned. The eyes, left to the preponderating forces, assume the direction seen during sleep and deep anesthesia—viz., divergence. Myopia is, therefore, frequently accompanied by divergent squint.

Sometimes individuals possess or acquire unusual power in developing one or other of these two functions. Thus, the hyperope may develop his accommodation sufficiently to equalize the disparity in the refraction and thus avoid squinting. The myope may also develop his convergence beyond the usual amount so as to prevent divergence. Hence all hyperopes do not have convergent squint; neither do all myopes have divergent squint.

2. *Inequality in the Vision of the Two Eyes, or Amblyopia of One Eye, Which Removes the Natural Stimulus of Diplopia to Exact Convergence.*—Amblyopia of the squinting eye is present in a large proportion of the cases of concomitant convergent strabismus, or, more accurately, the amblyopia of the squinting eye exceeds that of the other. Whether this amblyopia is a cause or a consequence of the squint has given rise to two theories. According to one theory, advocated by Donders and others, the squint causes the amblyopia which depends upon a loss of vision due to habitual suppression or to lack of use of the squinting eye—*amblyopia exanopsia*—or, according to Hirschberg's terminology, *amblyopia exablepsia*. According to the other theory, advanced by Schweigger, the amblyopia is a congenital defect which precedes and causes the squint. Priestley Smith points out that all eyes are amblyopic at birth, and reach the normal standard of vision only after several years. If strabismus is established before this standard is attained, further visual progress of the squinting eye is likely to be hindered or even arrested.

Ophthalmoscopically these amblyopic or so-called "neglected eyes" may be entirely normal, or there may be at times distinct changes in and around the nerve-head and in the macula. Central scotomas and contraction of the visual field are sometimes demonstrable, as the author

has shown, and in such circumstances these eyes are not susceptible of improvement in vision (see also page 496).

An amblyopia which removes the stimulus of diplopia to exact convergence may also include cases in which the visual acuteness is diminished by refractive differences in the two eyes, one eye being greatly inferior to its fellow by reason of a high degree of hyperopia or myopia, with or without astigmatism, by opacities in the media of one eye (especially corneal opacities), by congenital cataract, and by complete blindness. The failure to recognize diplopia causes the visual axes to vary considerably either toward convergence or divergence, without appreciation of this on the part of the patient. If the eyes are hyperopic, they are apt to converge; if myopic, to diverge. Numerous cases of squint exist without amblyopia, and the refraction of both eyes may be equal.

3. *Disturbances of Innervation and Defective Development of the Fusion Faculty.*—According to Hansen Grut, "convergent strabismus originates and continues as the result of an *innervation* which effects in the interni a shortening exceeding in amount that which is desirable. Divergent strabismus is the expression for a relaxation of convergence innervation, which permits the eye to take up its anatomic position of rest." According to Priestley Smith, "convergent strabismus is a disorder of innervation in which the visual centers fail to control the act of convergence, which is degraded and becomes automatic. It is excited by the act of accommodation and is excessive because uncontrolled. The failure of control depends largely upon faulty development of the visual apparatus. Hyperopia, when of considerable degree, predisposes to strabismus by demanding an abnormal effort of control. The disorder is confirmed and perpetuated by suppression of the function of the squinting eye."

According to Claud Worth, "when the fusion faculty is fairly well developed, neither hyperopia, anisometropia, nor heterophoria can cause squint. In fact, then, nothing but an actual muscular paralysis can cause an eye to deviate, in which case the resulting diplopia is intolerable. Sometimes, however, owing to a congenital defect the fusion faculty develops later than it should, or it develops very imperfectly, or it may never develop at all. Then, in this case, there is nothing but the motor coördinations to preserve the normal relative directions of the eyes, and anything which disturbs the balance of these coördinations will cause a permanent squint. Thus, the essential cause of squint is a defect of the fusion faculty." The provocation in the presence of this fundamental cause to squint may be supplied, he believes, by various conditions—for example, hyperopia, anisometropia, heterophoria, amblyopia of one eye, certain eruptive fevers and infections (whooping-cough, especially if hyperopia in any degree is present), violent mental disturbance, and hereditary influence. The influence of heredity in squint is an important matter, and Mr. Worth believes, and this certainly is in accord with the author's experience, that a history of heredity can be obtained in fully 50 per cent. of the cases. Therefore it is

important carefully to investigate early in life the eyes of children whose parents or grandparents have squinted. Recently Zentmayer has convincingly defended Donders' theory of the causation of strabismus (see paragraph 1, page 542).

A predisposition to strabismus may arise on account of the size and shape of the eyeball and orbit. A narrow, horizontal diameter of the face might predispose to convergent strabismus, or an unusually broad diameter to divergent strabismus. These conditions may coexist with hyperopia and myopia. A very short eyeball, flattened in its anteroposterior direction, by its greater facility of movement would render convergence easier; the opposite condition—namely, elongation of the anteroposterior axis of the eyeball—would render this movement more difficult. An unusual value of the *angle gamma* might create a disposition to squint by disturbing the relation between convergence and accommodation. At one time disparity in the length, thickness, and tension of opposing muscles was regarded as an important factor in the development of squint.

Single Vision in Concomitant Strabismus.—Diplopia is rarely noticed in concomitant convergent strabismus, because the deviating eye involuntarily suppresses the image, or else has learned to disregard it.

Suppression of the image is not, however, habitually permanent, or suppression does not extend over the whole visual field, and many patients can be made conscious of diplopia if a red glass or cobalt glass is placed before one eye and a prism before the other. When the squint is very large it may be necessary to correct the greater part of it with prisms before diplopia is manifest. If prisms and the red glass fail, Schweigger's test is as follows: A flame is placed to one side of and behind the squinting eye, and its image is thrown into this eye with a plane glass held close to it. When the reflex reaches the center of the pupil the patient sees it and can describe its relation to the image of another flame observed by the fixing eye at a distant point. With high degrees of amblyopia it may be impossible to produce diplopia.

In concomitant divergent squint, especially of low degree, and in the convergent strabismus of myopes, diplopia is not uncommon; also in moderate degrees of convergent strabismus and in the residual squint after tenotomy. Referring to the nature of diplopia in concomitant strabismus Claud Worth thus expresses himself: The subject of squint with diplopia sees with his deviating eye a faint eccentrically placed image of the object to which the fixing eye is directed, and suppresses the image of the object which lies in the axis of the deviating eye—*i. e.*, he sees two images of the same object, but not two different objects.

Sometimes after operation, as was first noticed by Von Graefe, the *diplopia* is anomalous or *paradoxic*, as it is called—that is, there is crossed diplopia with convergent squint. Javal observed and studied the same phenomenon in strabismic patients upon whom no operation had been performed. It has been explained on the theory that there has been developed in the squinting eye a spot identical with the macula

lutea of the straight eye, or that there has been developed what has been named a *vicarious fovea*. According to Tscherning, in certain cases of strabismus a period may be reached after operation when the patient localizes with reference both to the new and the old fovea, the result being *binocular triplopia*, a name given by Javal to the phenomenon. Verhoeff thinks paradoxic diplopia "is due not to the development of a new system of corresponding points, but to an absence of any such system whatever, so that when diplopia is produced, each eye localizes its image with regard to itself alone and hence more or less correctly."

Measurement of Strabismus.—1. Squint may be measured approxmately by the deviation inward of the pupil of one eye while the other eye fixes an object. The pupil being situated 10.5 mm. in advance of the center of rotation, its deviation inward or outward, measured on a rule, represents the tangent of the angle of the squint. A deviation of 1 mm. represents a squint of 5°. For this purpose an ordinary rule divided into millimeters may be employed, or a specially devised instrument curved to adapt itself to the curve of the eyeball and known as a *strabisometer*.

If diplopia is present, as Landolt has shown, it permits an accurate determination of the angle of strabismus. The procedure may be as follows:

> Upon a wall of the consulting room, in a horizontal line, and so as to be on a level with the eyes of the patient, who is about 3 meters from the wall, are permanently marked out tangents of angles of 5° each, as seen from the place where the squinting eye is. Exactly opposite to the squinting eye is 0°, while toward the right and left the points are marked up to 45° or more. The flame of a candle being held at 0°, and one eye of the patient being covered with a red glass, he is called on to indicate the position of the image belonging to the squinting eye, and the number on the wall which corresponds to this gives the angle of the strabismus.

In these circumstances the degree of prism necessary to fuse the double images may be used to measure the squint.

2. *Angular Method.*—The perimeter may be employed to measure squint with reasonable accuracy, although Worth condemns the method because it takes no account of the angle gamma. Landolt thus describes the method:

> The deviating eye, *R*, is placed at the center of the graduated arc of the perimeter, *P–P*, the arc lying on the plane of the deviation. The patient is then required to fix with *his two eyes* a distant object, *A*, situated at the central radius, *R–o–A*. This is the direction which the deviating eye should have in the normal condition. The point *n*, to which the eye in reality is directed, should now be determined; the angle *o–R–n*, formed by the deviating visual line *n*, with the normal line of fixation *A–o–R*, is the *angle of the strabismus*. In order to obtain this direction (*i. e.*, the point *n* at which the eye is directed) it would be necessary only to determine the visual axis. As this is not an easy matter, it is sufficient in practice to be contented with the optical axis; this differs from the former only by the angle gamma, which, in comparison with the large angle of the strabismus, may be neglected. The flame of a candle is moved along the arc of the perimeter until its reflexion is in the center of the pupil. This will occur when the flame is at *n*. The optical axis has now been found, and the size of the angle of strabismus may be read off.

Priestley Smith's Tape Method.—This is a very good method, although it is not very readily applied to young children. Worth describes it as follows: "A string 1 meter long has a ring at one end. To the ring is attached a graduated tape. The tape has a weight at its other end. The patient holds the free end of the string against his temple. The surgeon puts the ring on a finger of one of his hands, in which he holds an ophthalmoscope mirror. The tape is allowed to slide between the fingers of the other hand, the weight keeping the tape taut. The patient is first told to fix the mirror, while the light of a lamp is reflected into the fixing eye. The position of the image of the mirror on the cornea of the fixing eye is noted. The light from the mirror is now thrown on to the deviating eye, and the patient is directed to look at the surgeon's tape hand. This is moved horizontally till the position of the image of the mirror on the cornea of the squinting eye is similar to that which it formerly occupied on the cornea of the fixing eye. The string keeps the ophthalmoscope hand at 1 meter from the patient's eye. The observer keeps the tape hand as nearly as possible at the same distance from the patient's eye. The graduated scale on the tape, where it slides through the tape hand, shows approximately the angle of the deviation in degrees."

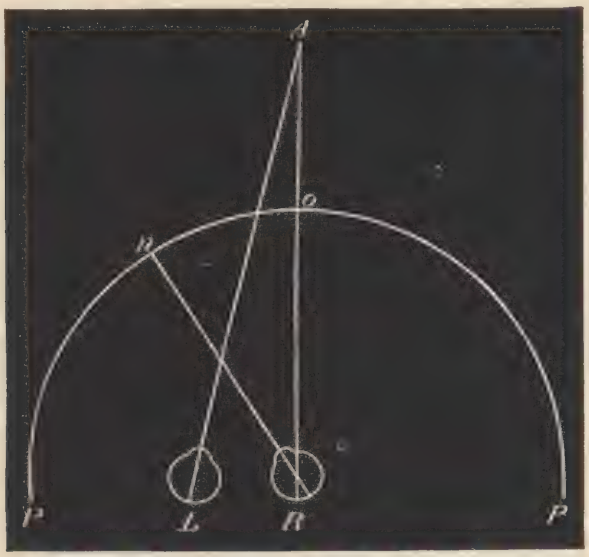


Fig. 239.—Measurement of squint with a perimeter.

Various forms of "deviometers" have been devised for measuring strabismus, especially by Nelson Black and by C. Worth.

Treatment of Concomitant Strabismus.—*A. Convergent Concomitant Strabismus.*

1. *Spectacle Treatment.*—Glasses which neutralize the refractive error should be ordered for every patient with convergent concomitant squint after the use of atropin has thoroughly paralyzed the function of the ciliary muscle. In the majority of cases the refraction is hyperopic and is often associated with considerable degrees of astigmatism. There is no difficulty in estimating exactly the proper lenses by means of retinoscopy, and if they are persistently worn early enough—before the fifth year—and, in addition, fusion-training is carried on, the strabismus

will be cured in a very considerable percentage (variously estimated from 30 to 70 per cent.) of the cases. It is important that this non-operative treatment of squint should be begun as soon after the discovery of the condition as possible, and glasses may usually be adjusted when the child is three years old; sometimes even earlier.

Prolonged atropinization of both eyes of very young children with squint, in order to remove the abnormal stimulus to convergence which results from overaction of the ciliary muscle, was at one time a much recommended method of treatment. As Worth points out, while it may produce temporary improvement, or even disappearance of the strabismus, it tends to increase the amblyopia of the deviating eye, and is, therefore, a therapeutic measure to be condemned. He properly recommends atropinization of the fixing eye only, so that the child shall acquire the habit of using the better (atropinized) eye for distant vision, and the poorer (unatropinized) eye for close vision, and thus avoid amblyopia from disuse. Suitable glasses should be worn. If the visual acuteness rises sufficiently, so that the originally deviating eye becomes the squinting eye, the drug must be discontinued, and, if the original condition repeats itself, be used carefully and intermittingly; for example, for a few days during each month (Worth). Recently Reber recommended invisible bifocal lenses in the treatment of esotropia in little children, a sphere of 2 to 3 D being added to the correcting lenses, the idea being "to set the spastic accommodation apparatus at rest." A 0.5 per cent. solution of atropin is used once daily for two months; at the end of this time it is discontinued. A similar recommendation has been made by Linn Emerson.

2. *Educative Treatment.*—This includes occlusion of the eye by means of a *shade* or *pad, bar reading, orthoptic training,* and *development of the fusion-sense.*

(*a*) *Occlusion of the Fixing Eye.*—The sound eye should be covered with a shade or bandage, not so much with the hope of improving the acuteness of vision of the deviating eye, but, as Priestley Smith has said, to compel it to use such vision as it has to promote fixation, and to prevent or stop the habit of suppression. If the child wears spectacles, as it should, a blinder of gutta-percha may readily be adjusted on the lens in front of the fixing eye. If the vision of the squinting eye is very imperfect, it is permissible during this treatment to wear the patch on this eye instead of the sound one for a few hours each day; but both eyes should not be allowed to be uncovered at the same time.

(*b*) *Orthoptic Training.*—This consists of the establishment of diplopia and training the eyes to fuse the double images, and is a method of treatment of squint which was especially advocated by Javal. It is particularly suited to moderate degrees of strabismus and to residual squint after operation. It requires considerable care and patience properly to carry out the details. In order to educate the fusion faculty the *stereoscope* should be employed. The patient's ametropia having been fully corrected, the exercises may be performed according to the method given by Landolt, as follows:

In an ordinary box-stereoscope, in the place of "views," two objects of some very simple shape are introduced—for instance, two vertical lines, one above and the other below the same horizontal line. These two lines, which may be brought toward or removed farther from each other at will, are placed at a distance about equal to that between the two eyes. In such circumstances their fusion into a single vertical line necessitates parallelism of the lines of fixation. This parallelism is generally possible only in the absence of any accommodative effect. Hence the sight-holes of the stereoscope are provided with + 6 D lenses (the length of the ordinary stereoscope being 16 cm.), which permit the subject to see at the distance of the objects without exercise of the accommodation.

The majority of patients do not succeed in fusing the images when their eyes are directed in a parallel direction. These latter generally show a certain convergence. The patient is then taught to find the distance between the two objects which is requisite for the fusion of their images. When this is accomplished, the two objects are gradually separated more and more in successive sittings until fusion is effected without the least convergence.

When binocular vision is obtained, with parallelism of the lines of fixation, which is equivalent to binocular vision at a distance, an attempt should be made to realize it for a point which requires a certain degree of convergence. To provoke a convergence of 1 meter-angle, the objects are brought together through a distance varying with the base line, the average being about 1 cm. In order to make the patient furnish an amount of accommodation equivalent to this amount of convergence, the strength of the convex lens is diminished 1 diopter. The trials are continued in this way until the two objects are brought on a vertical line. At this moment they require, for their binocular fixation, a convergence of 6 meter-angles and an accommodation of 6 D. An emmetrope would, therefore, have to remove the glasses from the stereoscope and see with the naked eye; an ametrope would require simply the correction of his refractive defect.

The illumination of the object looked at by the deviating eye may be increased in order to reinforce its visual impressions, as in Landolt's new stereoscope.

If the angle of squint is very great, both eyes cannot look at the same time into an ordinary stereoscope, and therefore a number of excellent instruments have been devised which can be adapted to the angle of squint. Kroll's orthoptic exercises, arranged by Perlia, which consist of colored plates placed in a suitable stereoscope, are useful. In many respects with the instrument devised by Claud Worth, to which he has given the name *amblyoscope*, the most satisfactory results are achieved, and, as this accomplished surgeon's method is now so much employed, the following directions have been written at the author's request by a member of his staff, Dr. H. Maxwell Langdon, who has devoted much attention to these exercises:

The amblyoscope consists of two tubes, one for either eye, each having its own illumination, which can be increased or lessened so as to equalize the visual impressions in case one eye is amblyopic. An object-slide is placed in the objective end of each tube, and is reflected in a mirror at the bend of the tubes, which is placed at the focal distance of convex lenses fitted in the proximal ends of the tubes, so that no accommodation is necessary. The proximal ends of the tubes are hinged in such a manner that they may be adapted to a convergent strabismus of 60° or a divergent strabismus of 30°.

The child's vision should be tested, if types or other signs cannot be utilized, with small white ivory balls, each with a diameter varying from $\frac{1}{2}$ to $1\frac{1}{2}$ inch. Each eye is tried separately, and the child is required to pick up the ball, which is

rolled with a twisting movement. If this test reveals that one eye is amblyopic and possesses one-sixth or less of visual acuteness, some form of blinder exercise should be instituted to improve the defective visual acuteness (see page 548). Preceding the exercises with the amblyoscope, the angle of strabismus should be measured according to the methods elsewhere described, the refractive error having been carefully and fully corrected, and the glasses being in position, and during all of these exercises the glasses must be worn. Amblyoscope training should be begun as soon as the child is old enough to look at ordinary pictures and to talk about them, because deviation yields far more readily to these exercises in young children than in older ones, and, moreover, after the sixth year it is usually practically impossible to make any satisfactory impression upon the defective fusion faculty. A child of three years is well able to take part in these exercises, especially if they are so conducted that they represent to him a game in which he may readily be interested. Usually one or two sittings a week, each occupying half an hour, are sufficient. The child should be seated on a chair between the surgeon's knees, and the angle of the tubes approximated to the angle of strabismus. Next, the illumination, which may consist of two electric-light bulbs, two lamps, or two candles, equally distant from each

Fig. 240.—The Worth-Black amblyoscope.

tube, are arranged, and an object-slide is placed in each holder. These object-slides should consist of pictures familiar to young children, but the ones used at first should be quite dissimilar; for example, the picture of a bird and the picture of a cage. The child is now required to look into the tubes, and is asked what he sees. If one eye is amblyopic to any considerable degree, it is probable that the image of the object before the better or fixing eye will be the only one which is visible. Hence, the illumination must be altered before the other object-slide can be seen by diminishing the light before the fixing or better eye, and increasing that before the amblyopic or squinting eye, continuing with this regulation of lights until both objects are visible and can be described by the child. This alteration in the lights can be accomplished in various ways; for example, by changing the distance of the lights, as Mr. Worth suggests, or by adapting to the amblyoscope, as the writer has done, a revolving wheel, which contains smoked lenses of different densities, and which can be turned before the non-amblyopic eye. Each object should be seen clearly, and the exercise should be varied with several pairs of object-slides. Next, the child is required to place one hand on each of the surgeon's knees and to tap that knee on the side on which the picture of the bird is seen. If the angle of the tubes is rapidly altered a position will be found where the slightest movement of the tubes causes the

picture of the bird to pass directly through from one side of the picture of the cage to the other. But, after continuing the exercises, the bird apparently will go directly into the cage, indicating that the child is acquiring a certain amount of fusing power. If one object is above the other, this vertical deviation must be overcome by means of prisms suitably placed in the grooved slides back of the focusing lenses. Dr. Nelson M. Black has added a vertical adjustment to the Worth amblyoscope,[1] which simplifies the correction of this deviation (Fig. 240). As soon as the child can easily merge the two objects, more difficult tasks are set, with slides demanding accurate and complete fusion, and by gradually widening the angle of the tube, a range of fusion which varies from 5° to 15° may be acquired by the patient.

Finally, a series of stereoscopic pictures, intended to teach the child the sense of perspective, are employed. During these exercises, by which the fusion faculty is stimulated and developed, the strabismus may do one of three things: it may disappear after a few days of training; it may gradually lessen; or it may not alter at all, and operative procedures are required to produce parallelism of the visual axes.

These methods to overcome the defective development of the fusion faculty should be faithfully tried in spite of the trouble which their use entails. Certainly the re-establishment of binocular vision in these circumstances is worth every effort.

(*c*) *Bar Reading* (*Controlled Reading of Javal*).—A pencil or, as Priestley Smith suggests, a thin strip of metal is held midway between the eyes and the book which they regard. Reading can then take place without interruption only if both eyes are employed. Priestley Smith describes the exercises as follows: "When the patient's fixing eye reaches that portion of the line which is hidden from it by the bar, he must use his other eye. Then the fixing eye is covered for a moment with a screen. Next, the patient is taught to occlude it for himself by a momentary closure of the lids. Soon he will be able to travel along the line with only a slight hitch where he closes the better eye, and at last he will read smoothly, keeping both eyes open." The method is chiefly effective when practised in conjunction with the use of the shade—that is, the shade covers the fixing eye and it is uncovered only for the purpose of bar reading; and this should be practised as much as possible. Indeed, according to Javal, the exercises must be continued for months, but there seems no doubt that they are efficient aids in the recovery of binocular vision. It need hardly be stated that the exercises are not suited to very young children. They are valuable in the residual squints after operation.

3. *Operative treatment* consists of tenotomy of one or both internal recti, with or without advancement of the externi, or of bilateral advancement of the externi without tenotomy of the interni (see page 671). If possible, operation should not be undertaken until the fusion faculty has been developed by the exercises already described, and in no cir-

[1] The author uses, with much satisfaction, Dr. A. Maitland Ramsay's (The Ophthalmoscope, January, 1905) modification of Worth's amblyoscope, in which totally reflecting prisms are employed instead of mirrors. Back of each picture is placed a small electric lamp, the relative brightness of which can be varied to any desired extent by shifting a key, which increases the resistance for one of the lamps while it diminishes it for the other. The author has slightly modified this instrument by adding to it an arrangement by which prisms to correct vertical deviations may be inserted, and scales which indicate the exact separation of the tubes to suit the interpupillary distance and the degree to which the tubes must be converged or diverged, according to the character and angle of the squint.

cumstances until the refractive error has been fully corrected and glasses have been worn for at least six months. If, in spite of such treatment, the deviation remains constant, operation is necessary, and may be performed if the child has passed the sixth or seventh year. If the exercises have failed to develop the power of binocular fusion, or if these exercises have begun at a time too late to expect this result, it would seem wise, under most conditions, as E. Jackson insists, to wait until the patient has reached an age when the operation can be performed under local anesthesia and intelligent co-operation secured; to wait, in short, until after the period of rapid growth and development. Worth, on the other hand, has no objection to general anesthesia.

There is much difference of opinion in regard to the operations which should be practised for the relief of convergent strabismus, and each case must be carefully studied before a correct decision can be reached. The practice, at one time almost universal, of endeavoring to correct convergent strabismus by means of tenotomy of one or both interni, according to the amount of the deviation, has been largely and very properly abandoned in favor of advancement of the externi.

While it is true that in small squints (15° to 20°) tenotomy of the internus of the deviating eye, if this is not seriously amblyopic, will often yield, temporarily at least, a satisfactory cosmetic result and that in *alterating squint*, with good vision of each eye, even a double carefully performed tenotomy has been recommended, in most circumstances a tenotomy should be avoided. As Landolt has well said, the "dosage" of tenotomy is uncertain, and from the dynamic standpoint its effects are unfavorable. The outward rotation tends to increase, the palpebral fissure is widened, the eye often is slightly prominent, and inward rotation is permanently weakened. Free division of the tendinous insertion of the interni and the surrounding capsular attachments is *never permissible*, and almost sure in subsequent years to lead to divergence; indeed, this may be the result of bilateral tenotomies of the interni, even if they are carefully performed. Admirable results follow bilateral advancement of the interni close to the cornea (tenotomy is not performed), and in pronounced squint this may be combined with resection of more or less of the muscle. In this respect the author can confirm the value of Landolt's advice and method, and is in full accord with Claud Worth's condemnation, under most conditions, of tenotomy. In slight degrees of squint simple advancement will usually suffice. After operation either both eyes should be bandaged until the sutures are removed, or both eyes should be unbandaged and the patient from the first directed to wear his correcting glasses.[1] (For the methods of performing tenotomy and advancement, see page 671.)

B. Divergent Concomitant Strabismus.—The treatment of this form of concomitant squint includes the correction of the error of refraction with suitable glasses, training convergence, and operative measures.

[1] For an important paper on this subject, the reader is referred to "The Indications for Operating in Heterophoria and Squint," by Alexander Duane, Archives of Ophthalmology, vol. xl., No. 4, p. 390, 1911.

(*a*) *Glasses* which neutralize the refractive error (most commonly myopia or myopic astigmatism) should be adjusted according to the rules which are given in the chapter devoted to the measurement of abnormal refraction. H. Landolt recommends concave glasses of such strength that they overcorrect the myopia. According to him the excessive effort of accommodation stimulates convergence. Moderate degrees of divergent deviation may often be favorably influenced by prismatic exercises (see page 559).

(*b*) *Operative measures* depend entirely upon the degree of the deviation, the vision in the diverging eye, and the cause of the difficulty. When true divergent strabismus exists, it is usually necessary to perform an operation to correct it. This may be either tenotomy of one or both externi, or this operation may be combined with advancement of the internal rectus. Usually advancement of the interni, one or both, according to circumstances, is preferable to tenotomy of the externi; but the latter operation may be needed to secure parallelism of the visual axes. A coexisting vertical deviation should be remedied, and some operators (Hansell and Reber) prefer to make the vertical adjustment before attacking the lateral deviation.

Vertical Strabismus.—Often there is an *apparent* vertical deviation associated with lateral strabismus, which disappears with the correction of the latter defect. In cases of *true* vertical deviation requiring operation Worth recommends advancement of the inferior rectus muscle of the eye which turns upward. Landolt, although opposed to tenotomy of the lateral muscles, does not object to this operation on the superior rectus. All surgeons agree that tenotomy of the inferior rectus is not permissible. In certain circumstances an upward deviation is due to spasmodic action of the inferior oblique and must be corrected by tenotomy of this muscle (see page 672).

Results of Operation in Convergent Strabismus.—The effect of the operation, if well performed, is to produce parallel visual axes, and thus remove the disfigurement. Properly speaking, a *cure* is obtained only when there is improvement in the vision of the squinting eye and binocular vision is secured. There has been much difference of opinion on this subject, and some authors—for example, Lang and Barrett—have questioned if valuable improvement in the vision of the amblyopic eye ever takes place. Noyes believed that binocular vision was secured in less than 20 per cent. of the cases, even after the most perfectly performed operations and after the patients' eyes had been carefully corrected with glasses and trained by orthoptic exercises. Careful training of the fusion faculty should begin early and the exercises already described should be systematically carried out. Indeed, as may have been inferred, the necessity for operation, if only the educative treatment of strabismus is begun soon enough, is sure to diminish, but should an operation become necessary, it is also sure to be followed by far better results than can be achieved in the absence of such training.

It is often difficult to ascertain whether true binocular vision exists, especially in young children, and successful bar reading, usually quoted

as a sufficient test, is, according to Priestley Smith, not without its fallacies. This author tests as follows:

A reversible frame carrying red and blue glasses is placed in front of the patient's spectacles, and he is shown, at the reading distance, a card with three disks on a black ground, a white one in the middle, a red one above, and a blue one below. If he can see all three at once and in a line, he is probably using both eyes and fusing the two images of the white disk. If with each eye alone he sees two, but with both eyes three, the proof is fairly positive. The test may be improved by placing a black letter on each disk, which, if the patient has sufficient vision, he should read. The same test with larger objects may be used at longer ranges. The light should be good, but not too strong, and not artificial. Hering's drop-test may also be employed. A simple and ingenious diaphragm test for binocular vision has been devised by N. Bishop Harman.[1]

Spastic Strabismus.—This condition, more properly characterized by the term *convergence cramp*, or *spasm*, is seen in hysteria, and is characterized by convergent squint, limitation of the motility of the external recti, and by homonymous diplopia. It somewhat resembles paralysis of the abducens, for which it may be mistaken, but from which it should be differentiated by a study of the double images. Sometimes this form of strabismus or, rather, convergence spasm is accompanied with other hysteric manifestations—blepharospasm and ptosis—and may be a symptom of meningitis.

Abnormal Balance of the Ocular Muscles, or Heterophoria (*Latent Deviation*).—This is, as already defined, a disturbance of the normal balance of the exterior eye muscles, which creates a tendency for the visual lines to depart from parallelism, a tendency which is checked by the habitual desire for binocular vision, or that vision in which the images of an object formed on the retinas of the two eyes make but a single mental impression.

Heterophoria (*imbalance*, according to Gould) differs from *squint* or *heterotropia* because in the latter the fusion of the images is usually impossible—*i. e.*, binocular single vision is absent—and there is an evident departure of the visual lines from parallelism, which gives rise to the term which designates the condition.

Causes.—Imbalance of the ocular muscles may be due to: (*a*) weakness of the muscles (properly called insufficiency) of congenital origin, or depending upon a general lack in muscular tone, the result of anemia, nervous exhaustion, pelvic disorders, etc., or malaria, rheumatism, gout, etc., diseases which, however, may also be potent by affecting not the muscles themselves, but their innervation; (*b*) errors of refraction and disturbance of accommodative efforts (*accommodative heterophoria*); (*c*) the anatomic arrangement of the parts—for example, faulty attachment of the muscle (*concomitant heterophoria*); (*d*) excessive action or spasm of opposing and dominating muscles (*spasmodic heterophoria*); (*e*) disturbances of innervation (*central heterophoria*); and (*f*) a paretic condition of the muscle (*paretic heterophoria* of Duane).

[1] For description, see Ophthalmic Review, vol. xxviii, 1909, p. 93.

Varieties.—According to Stevens' nomenclature, if there is a tending of the visual lines in parallelism, the term *orthophoria* is applied; if there is a tending of these lines in some other direction, the term *heterophoria*. Heterophoria is divided into: *esophoria*, a tending of the visual lines inward; *exophoria*, a tending of the visual lines outward; *hyperphoria* (right or left), a tending of the right or left visual line in a direction above its fellow. *Cyclophoria*, according to Savage, is a want of equilibrium on the part of the oblique muscles (see also page 76).

Abnormal inward tending of the visual lines may depend upon excessive convergence or deficient divergence, or upon these conditions combined. Duane[1] describes the signs as follows: If *esophoria* for distance is less than for near, abduction (prism-divergence) not disproportionately low, adduction (prism-convergence) readily performed, esophoria marked at the near point and the convergence near point excessive, *convergence-excess* is present. If esophoria for distance is much greater than for near, abduction (prism-divergence) disproportionately low or absent, adduction (prism-convergence) normal or subnormal, esophoria slight, absent, or replaced by exophoria at the near point, and the convergence near point not abnormally close to the nose, *divergence-insufficiency* is present.

Convergence-excess is followed, if of long standing, by divergence-insufficiency, and similarly divergence-insufficiency by convergence-excess. In the *mixed form* thus produced there are marked esophoria for near and far, excessive approximation of the convergence near point, and limited, absent, or negative abduction (prism-divergence). Finally, the deviation ceases to be latent, binocular vision is lost, and *esophoria* passes into *esotropia*.

Abnormal outward tending of the visual lines may depend upon deficient convergence or excessive divergence, or upon these conditions combined. Duane records the symptoms as follows: If *exophoria* for distance is slight or absent, abduction (prism-divergence) not very great or even subnormal, adduction (prism-convergence) exceedingly difficult, exophoria marked at the near point, and the convergence near point less than 3 inches and maintained only for a moment, there is *convergence-insufficiency*. Sometimes this may be so great as to constitute a *convergence-paralysis*. If exophoria for distance is marked, abduction (prism-divergence) is high, adduction (prism-convergence) normal or not greatly subnormal, and the convergence near point normal, there is *divergence-excess*.

Convergence-insufficiency is followed, if of long standing, by divergence-excess, and, similarly, divergence-excess by convergence-insufficiency. In the *mixed form* thus produced there are marked exophoria for near and far, excessive abduction (prism-divergence), and marked retreat of the convergence near point. Finally, the deviation ceases to be latent, binocular vision is abandoned, and *exophoria* passes into *exotropia*.

[1] American Text-book of Diseases of the Eye, Ear, Nose, and Throat, edited by de Schweinitz and Randall, page 515. The descriptions which follow are condensed from Duane's article.

If hyperphoria, again to quote Duane, varies noticeably in different directions of the gaze, it is *non-comitant* and is due to underaction or overaction of one or more of the elevators or depressors; it may be due to spasmodic action of these muscles and may spontaneously disappear. If hyperphoria remains the same in all directions of the gaze, it is *comitant*, and may be due to excessive sursumvergence, or more frequently to the same agencies which produce non-comitant hyperphoria which has become comitant.

Whether the hyperphoria is due to overaction or underaction of one or other set of muscles may be determined by examining the rotations of the eyes (see page 522). Excessive upward rotation would naturally indicate overaction of the elevators of the hyperphoric eye, and excessive downward rotation overaction of the depressors. Deficient upward or downward rotation would indicate underaction of the vertical muscles, and in these circumstances diplopia is readily elicited, as it is in paretic conditions, by carrying the test-light in the direction of the action of the affected muscle. Hyperphoria usually does not tend to increase, and therefore binocular fixation is usually retained, and it is comparatively rare for *hyperphoria* to pass into *hypertropia*.

Full correction of hyperopia disturbs the relative range of accommodation and convergence and may cause exophoria (convergence-insufficiency—relative insufficiency of the interni, according to Risley). The same condition is seen in myopes who do not use glasses at close ranges and in presbyopes whose reading-glasses are too strong. Suitable glasses, or a modification of the glasses, and sometimes exercises with prisms, will relieve the condition (see also page 150).

Relative Frequency of Heterophoria.—Faulty directing power of the vertical muscles (hyperphoria) is usually stated to be the least common of these anomalies, but is much more frequent than was once supposed, and, according to Hansell and Reber, will be found in one-third of the cases of refractive anomalies. Many of these hyperphorias, however, are temporary in character and require no treatment except correction of the refraction and any underlying constitutional condition. The power of hyperphoria in causing asthenopic symptoms is of paramount importance, and, according to Stevens, its rôle in disturbing the action of the lateral muscles is significant.

It has been stated exophoria is the most common type of muscular imbalance, but Noyes considered esophoria more frequent than exophoria, the preponderance being 3 to 1, and this certainly is in accord with common experience. Heterophoria may be associated with any type of refractive error.

Difference Between Heterophoria and Heterotropia (Squint).—The essential difference between these two conditions has already been several times defined, and the passage of a heterophoria into a heterotropia has been described. The differential diagnosis should depend upon the results obtained from the application of certain tests. Duane describes these as follows:

"If there is any noticeable deflection behind the screen (see page 72),

the *screen-test is applied in a second way* or by *binocular uncovering.* This procedure consists in covering the left eye and then uncovering both eyes and noticing the movement that takes place. If, on thus uncovering the left eye, the right eye remains steady and the left moves into position, the patient has binocular fixation, and the deflection was a heterophoria and not a squint. If, however, the right eye should move out of its position and the left eye should move into place, there is a squint and the left is the fixing eye. If neither eye moves, there is a squint and the right is the fixing eye. By repeating this experiment with each eye alternately the examiner can tell whether there is a habitual binocular fixation, an alternating fixation, or a uniocular squint. The diagnosis between the three may be conveniently formulated as follows:

"1. If in binocular uncovering *but one eye moves*, heterophoria and not squint exists.

"2. If either *both eyes move* or, in spite of there being an evident deviation, *both eyes remain steady*, squint exists.

"3. In the latter case, if, when the left eye is uncovered, the eyes behave in the same way as they do when the right eye is uncovered (both alike moving or both alike remaining steady, no matter which eye is uncovered), the squint is *alternating.*

"4. If, when one eye—for instance, the right—is uncovered, both eyes move, and when the other eye (in this case the left) is uncovered both eyes remain steady, the squint is *uniocular* (confined in this case to the left eye)."

Symptoms.—These are usually classified under the general term *muscular asthenopia*, and may be divided into the *ocular* and the *general* symptoms.

To the *first group* belong pain, often over the insertion of the affected muscle, and especially marked when the eye is suddenly moved in the direction of its action; blurred vision and imperfect power of working at close ranges; inability to gaze attentively at a stationary object or person even at long ranges, and great discomfort when attempting to watch moving objects; dread of light and blepharospasm, often confined to a few fibers of the orbicularis; and local congestions of the conjunctiva, especially over the insertion of the muscle, and on the margins of the lids. Often there are eccentric poses of the head, distortions of the features, especially wrinkling of the forehead, contractions of the sternocleidomastoid, and tilting of one or other shoulder.

In the *second group* the prominent symptom is *headache*, which may be situated in any portion of the cranium, but which is common in the occiput. The pain may immediately follow the use of the eyes, or be delayed, or come on at a certain hour of the day, or even night. The headache may assume the migrainous type.

Pain in the back, especially between the shoulder-blades, or precordial pain, is common. Vertigo, generally subjective, is frequent, one variety being characterized by a sense of falling forward when walking in a crowd, associated with confusion of ideas. Drowsiness and, on the

other hand, insomnia may be present, and a variety of general or so-called reflex neuroses.

Chorea, epilepsy, pseudochorea, night-terrors, melancholia, neurasthenia, hysteria, palpitation of the heart, indigestion, constipation, flatulent dyspepsia, and a host of other complaints have been attributed to muscular imbalance, and also to accommodative strain, and under these conditions the eyes should always be examined and the ocular defects corrected (see also page 141). Many instances of remarkable nervous disturbances are associated with heterophoria, especially hyperphoria (as well as with refractive error), and cure will often follow the relief of the ocular difficulty. Unfortunately, the whole matter has not always escaped exaggeration.

Method of Examination.—The method of examining the ocular muscles has been fully described on pages 72–78. (See also Appendix, page 690.) Two points deserve reiteration—viz., that a measurement of the relative weakness and power of the muscles is inexact unless this has been made after the refractive error has been corrected, and the muscles have been tested through the correcting lenses; and that the examinations of the muscles should be made both for the *near* and the *far* point—*i. e.*, at 30 cm. and 6 meters—the latter being the more important determination.

Treatment.—As Duane has well said, "There must be no attempt to treat an insufficiency simply as an insufficiency, but account must be taken of the complex causes which lie at the root of it."

Strict orthophoria is rare. Small errors of the lateral muscles are often unimportant.

If there is a constitutional disorder or an insufficient nervous tone, this must be treated on general principles. Strychnin or ascending doses of tincture of nux vomica have been recommended. Galvanism may be tried, but it is doubtful if the current reaches the muscle. Large doses of tincture of hyoscyamin are of distinct advantage in cases of spasmodic heterophoria.

In every case of heterophoria the refractive error should be corrected according to the rules already laid down. In many instances this alone will suffice to restore the balance and cure the asthenopia. In esophoria of accommodative origin the total amount of the hyperopia and astigmatism should be neutralized with suitable glasses, which are to be worn constantly, and in high degrees of this muscle defect a convex sphere of from 1 to 2 D may be added to the distance correction and used in near work in order to diminish accommodative overactivity; in exophoria, especially with insufficiency of convergence, the full correction of the myopia should be ordered (compare with page 136). Where esophoria exists with myopia and exophoria with hyperopia, this plan must be modified, and an undercorrection of the refractive error prescribed. Convergence-insufficiency caused by glasses of improper strength has been described on page 556.

If the symptoms continue, recourse should be had to *gymnastic*

exercises with prisms. The object is to strengthen abduction and adduction. A number of methods are in common use:

1. The patient is instructed to practise fusing the double images produced by viewing a candle-flame situated 6 meters away. Abduction (prism-divergence) and adduction (prism-convergence) are exercised, beginning with the weakest prisms and gradually increasing to the strongest. This plan probably acts, as Maddox suggests, by training the efforts of accommodation and convergence to assume broader relations to each other in their work. It is efficient in selected cases.

2. Rhythmic exercises, contraction and relaxation of the muscle being secured by causing the patient to view a small gas-jet 20 feet distant through *adverse prisms*,[1] which are lowered and raised at regular intervals of five seconds, beginning with weak and gradually going to stronger numbers. This is the method of Dr. G. C. Savage. This author also recommends rhythmic exercises by rotating convex cylinders before the eye for the relief of *cyclophoria.*

3. The patient is provided with prisms double the primary distant adduction-power. The candle-flame is then slowly carried, while he regards it fixedly and continuously from the near point to the distant point. This is repeated until, without difficulty, he can, through the prisms, secure a single image in all parts of the room. The strength of the "handicap-prisms" should be gradually increased. For esophoria the reverse of the plan is pursued. This is the method advocated by Dr. Gould.

Referring to muscle exercises, Duane states that he regularly employs four, namely: distant exercise with prisms, bases out; exercise with prisms, bases out, at near points; exercise with prisms, bases in, at near points, and exercises in converging on a pencil-point (see also page 74). Exercises with prisms, bases out, are followed by most satisfactory results in exophoria, especially in convergence insufficiency, and should always be practised not only at the distance, but, as Duane insists, at the near point. Exercises with prisms, bases in, in esophoria at the distant point, in the author's practice, have not been of any value, but recent experience, based on Duane's advice to use diverging prisms at near points in cases of convergence-excess, indicates that the method may produce good results. The author has failed to observe relief in hyperphoria from prismatic exercises, but Savage's method has received the commendation of many competent observers, and should be tried.

The next method of treatment is the *prescription of prisms.* The action of prisms has been explained (see page 18). Much difference of opinion exists in regard to their therapeutic value. The author believes with Duane that "the employment of prisms in lateral deviations is to be avoided except as a temporary measure, since prisms, base in, tend to produce convergence-insufficiency, and prisms, base out, convergence-

[1] "Adverse prism" is a term used by Maddox, and means one with its apex set in the opposite direction from a "relieving prism"; for example, base out if the interni are to be affected, base in if the externi are to be exercised.

excess, so that in both cases they ultimately increase the deviation which they are designed to correct." Prisms may be ordered when the range of movement is perfect but in an unavailable position. The base of the prism should be placed toward the muscle which is to be aided, and the apex toward the muscle which is to be weakened.

It is usually uncomfortable for the patient to wear more than 4° or 5° constantly—*i. e.*, 2 or $2\frac{1}{2}$ over each eye. This statement, however, admits of many modifications, and often the strength of the prism may be increased much beyond this limit.

In permanent latent deviations of the vertical muscles (right or left hyperphoria) the defect is often quite small, and usually not above 4° or 5°; hence prisms may readily be ordered for continuous use, and combined with the lenses which correct the refractive error, forming a *prismosphere*. If, for example, there is right hyperphoria of 2°, a 2° prism base down before the right eye corrects the difficulty, or, what is equivalent, the prism may be divided between the two eyes—*i. e.*, 1° base down before the right, and 1° base up before the left. It is safe to correct very trifling errors in the vertical muscles either with prisms or by decentering the correcting lens to an equivalent degree (see page 20), providing these errors are still maintained after continuous use of glasses which neutralize the refractive error.

In esophoria, which is a frequent cause of muscular asthenopia, prisms are often combined with the correcting lenses and worn constantly. For the reasons before stated, the author doubts the value of constant prisms, with rare exceptions, under these conditions.

In exophoria the constant use of prisms is not advisable. On the other hand, they may be a great help in relieving the strain upon convergence by removing the point of intersection of the visual axes farther from the eyes, and for this purpose they are combined with reading-glasses. In high degrees of exophoria, or if there is actual divergence, abductive prisms are of little use; if the deficiency of the directing power is determined to be equivalent to 10°, one-half of this may be corrected—*i. e.*, $2\frac{1}{2}$° base in over each eye; if it is desired to remove all effort, the faulty tendency is measured in the usual way, and if it is within suitable limits, prisms are ordered, combined with the correcting glasses which neutralize the defect.[1]

It has also been suggested to strengthen the muscles by means of *orthoptic exercises*—*i. e.*, by causing them to make forced movement in different directions; by making forced movements of convergence, the patient being required to look at near objects—"thumb exercises"; by requiring the eyes to unite the images of two slightly separated objects. Stereoscopic exercises are also of advantage.

In the event of failure to relieve asthenopic symptoms by the methods thus far described operative procedure may be necessary. This consists of partial, complete, or graduated tenotomy of the an-

[1] When a spheric lens is combined with a prism, the deviating effect of the combination is different from that of the prism alone. Mr. Archibald Percival (Ophthalmic Review, October, 1891) has constructed elaborate tables which give the deviating effect.

tagonistic muscle, or of advancement of the feeble muscle (see chapter on Operations). Whether advancement or tenotomy should be performed depends upon the conditions. Advancement is indicated to strengthen a weak muscle, and tenotomy if an overstrong muscle is to be weakened. For example, in exophoria due to convergence-insufficiency advancement of the internus is a more rational procedure than tenotomy of the externus, but if the exophoria depends upon divergence-excess, tenotomy of the externus is the better operation. The same advice applies to esophoria, convergence-excess indicating tenotomy of the internus, and divergence-insufficiency advancement of the externus. According to circumstances, one or both externi or interni may need readjustment or division. Operations on the vertical muscles must be governed by similar rules. In cases of insufficiency Landolt performs advancement on one eye. Surgical interference is required only after all other measures have been long and faithfully tried and have failed to give relief. While cases of muscular imbalance, best treated by operative interference, are encountered (aptly called by Risley "absolute insufficiencies," equivalent to the structural and insertional anomalies of Duane), in the opinion of the author they represent a limited proportion of the whole number. Moreover, as our knowledge of the etiology of abnormalities of muscular balance increases and our methods of non-surgical treatment improve, this number grows steadily smaller.

So-called graduated tenotomies and partial tenotomies are performed by some surgeons, and it is asserted that adjustments are exactly made, but in them the author has no faith. It is true that brilliant results have been made and described by experienced operators, but there is no doubt that a good deal of injudicious "snipping of the tendons of the ocular muscles" has been practised.

Nystagmus.—This term is applied to a condition characterized by an involuntary, rapid movement of the eyeballs. The movement may be lateral, vertical, rotary, or mixed—*i. e.*, a compound of two varieties. It has been especially studied in this country by Duane and Percy Fridenberg.

The condition may be *congenital* or *acquired*, and is bilateral in the vast majority of cases, although a few instances of unilateral nystagmus have been reported, with the movements usually in the vertical direction. It is possible, however, inasmuch as slight forms of nystagmus are detected only by using the ophthalmoscope and watching the fundus, that some of these supposed unilateral cases have actually been bilateral.

The movement is nearly always in the lateral direction. According to Gowers, the extent varies from 1 to 10 mm., and the frequency from 60 to 200 separate oscillations a minute.

Congenital nystagmus is seen with cases of defective construction of the eyeball—coloboma, microphthalmos, etc. It is also common in albinism and in color-blind persons with small central scotomas (C. L. Franklyn). Nystagmus also occurs with opacities of the media,

especially when such obstruction to the rays of light has been caused by diseases occurring early in life and in blind eyes. In blind eyes the so-called *searching movements* occur; that is, the eyes make a comparatively slow and wide movement from the primary position, to which after a time they again return, and so the movements are repeated.

Hereditary nystagmus, extending through a number of generations, has been especially studied by Nettleship: two types exist, the first is male-limited, the transmission being through unaffected females; in the second both sexes are affected, the descent being most frequent through the females. In this class head movement is common.

Nystagmus may be acquired in the pursuit of certain occupations, especially mining, and is commonly known as *miners' nystagmus*. It generally occurs among those who use a dim light, and whose work necessitates keeping the eyes in an unusual position for many hours together (Snell). T. L. Llewellyn's studies lead him to believe that the chief cause of this form of nystagmus is strain caused by deficient light. Errors of refraction increase the liability to nystagmus. Miners' nystagmus has also been attributed to muscle fatigue and to insufficiency of the fusion power (Dransart and Van Houtte). The visual fields, according to Cridland, are similar to those observed in traumatic neurasthenia.

Finally, nystagmus or, more accurately, pseudonystagmus is exceedingly common in diseases of the nervous system, particularly disseminated sclerosis and Friedreich's ataxia. It occurs in many diseases of the brain, and has been noted with great frequency in tumors of the cerebellum. Head-jerkings, associated with nystagmus, may occur in young children, constituting the so-called *spasmus nutans*. It may be unilateral.

Nystagmus has been ascribed to chronic fatigue of the muscles and oscillation of the globe consequent upon the muscular atony, and also to a central origin. Duane believes that true nystagmus depends upon a perversion of the centers for parallel and parallel-rotatory movements and not on peripheral muscle or nerve lesions. *Labyrinthine nystagmus*, produced by irritation of the labyrinth, may be horizontal, vertical, and rotary, and the movement consists of a slow followed by a rapid oscillation, most intense when the visual axes are directed toward a certain point on the limits of the binocular field of vision (A. W. Ormond, Sydney Scott).

Treatment.—If practicable, in cases of nystagmus where there is interference with the reception of perfect retinal images, the best possible vision should be restored by correction of refractive error, by tenotomy, or by iridectomy for new pupil, according to the indications. Very often good results have been noted. If nystagmus is brought about by any occupation, the evident indication is to remove the patient from his surroundings. For central nystagmus from brain or cord disease there is practically no remedy. In some instances of acquired nystagmus benefit has been reported from the local use of eserin and the internal administration of strychnin.

Monocular Diplopia.—This character of diplopia has been explained by one of several conditions: (1) By anomalies of refraction, particularly astigmatism; (2) by opacities in the cornea or lens or by anomalies of the pupil, for example, polycoria (see also page 395); (3) by irregular cramp of the ciliary muscle; (4) by complete or partial constriction of the eyelids, by which they are made to impinge on the cornea (G. J. Bull); (5) by hysteria or allied functional nervous disturbance; (6) by organic disease of the brain or its membranes, associated with abducens paralysis (Gunn and Anderson); (7) by simulation, the symptom being an invention of the patient for the purpose of magnifying the result of injuries.

CHAPTER XX

DISEASES OF THE LACRIMAL APPARATUS

Diseases of the lacrimal structures naturally divide themselves into those which have their seat in the lacrimal glands and those which affect the drainage system—*i. e.*, the puncta, canaliculi, lacrimal sac, and nasal duct.

Dacryo-adenitis.—This is an inflammation of the lacrimal gland, a comparatively rare affection, which may be *acute* or *chronic*, *suppurative* or *non-suppurative*.

Non-suppurative dacryo-adenitis, on account of its analogy to bilateral parotitis, has been called *mumps of the lacrimal gland* (Hirschberg). It may be caused by influenza, small-pox, leukemia, and mumps. Tuberculous dacryo-adenitis is rare, according to Stieren, who reports an example of this affection, only 12 cases being on record. The unilateral chronic form of inflammation of the lacrimal gland is more common, and has been observed in scrofulous subjects, and may be caused by an injury or follow diseases of the conjunctiva and cornea.

If the gland is chronically enlarged, palpation will reveal its lobulated border; if the inflammation is acute, there are pain, tenderness, and swelling at the upper and outer part of the eyelid, with chemosis of the conjunctiva. This may go on to suppuration, and the abscess usually points upon the skin, but occasionally through the conjunctiva. Acute dacryocystitis may result from infections from the conjunctiva, from infectious diseases, and from injury. *Metastatic dacryo-adenitis* in the subjects of gonorrhea has been described.

Treatment.—Warm applications and poultices to relieve pain are needed, and at the first appearance of pus an incision should be made either through the integument parallel to the eyebrow, or through the conjunctiva. If induration of the gland occurs, this should be treated locally with iodin or iodid of cadmium ointment.

Hypertrophy of the lacrimal gland has been observed at birth, but usually is seen in later years, and consists in an indurated lobulated tumor having its situation in the upper and outer part of the orbit.

Atrophy of the lacrimal gland, as the result of xerophthalmos, has been described.

Spontaneous prolapse of the lacrimal gland appears in the form of a soft movable tumor under the upper eyelid. Hypertrophy and prolapse or prominence of the palpebral portion of the lacrimal gland may occur in various corneal and conjunctival inflammations, and is evident on everting the upper lid.

The **treatment** consists of extirpation of the prolapsed organ.

Traumatic Dislocation of the Lacrimal Gland.—This is a rare accident, and occurs most frequently in young children (Villard), but also in adults (E. Jackson). Usually the gland prolapses through a wound in the upper lid. If the gland can be returned to its place and the wound sutured, this procedure is preferable to excision, which, however, may be necessary, especially if infection occurs.

Fistula of the Lacrimal Gland.—This may remain on account of the rupture of an abscess, but has also been recorded as a congenital defect at the outer third of the upper lid. A fringe of hair may surround the opening of the fistula.

The fistula may be closed by repeated cauterization or by a plastic operation; in the event of the failure of these measures, extirpation of the gland is indicated.

Syphilis of the Lacrimal Gland.—The lacrimal gland is singularly free from syphilitic affections, but specific induration and inflammation have been described, and in any instance of unaccounted hypertrophy of this gland, careful antisyphilitic treatment should be undertaken before resorting to surgical measures.

Dacryops.—This affection, often classified with diseases of the conjunctiva, is caused by a cystic distention of one of the main gland-ducts or of one of those of the accessory lacrimal gland, and appears in the form of a bluish, translucent swelling beneath the conjunctiva at its upper and outer part. If the mouth of the excretory duct is not occluded, pressure upon the tumor causes a few drops of liquid to escape. It is a comparatively infrequent affection, about 35 cases being on record (Ernest Thomson).

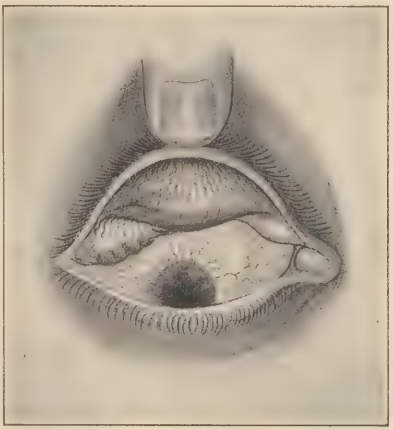


Fig. 241.—Enlargement and prolapse of the palpebral portion of the lacrimal gland in an eye with kerato-iritis.

Tumors of the Lacrimal Gland.—Adenoma, fibroma, myxoma, adeno-angioma, epithelioma, carcinoma, osteochondroma, lymphoma, cylindroma, and sarcoma occur. Tubercle, in the form of a small almond-shaped tumor, has also been reported in this region. Cysts and concretions (dacryoliths) occur. The concretions are contained in the excretory ducts, and are composed of concentric, chalky masses (Levi). According to Warthin, the majority of lacrimal tumors are, most probably, mixed tumors of endothelial origin, similar to those of the parotid and submaxillary glands. They tend to form cartilaginous, hyaline, and myxomatous tissue, and their malignancy is relatively slight. F. H. Verhoeff believes, however, that mixed tumors of the lacrimal gland are essentially epiblastic in origin, that they are dangerous to sight and to life, and that they should be extirpated as soon as possible.

To a symmetric enlargement of the lacrimal and salivary glands the name *Mikulicz's disease* is applied. It is unassociated with any systemic affection. According to S. Lewis Ziegler, the tumefactions should be regarded as true lymphomas.

Anomalies of the Puncta Lachrymalia and Canaliculi.—1. **Congenital Anomalies.**—Double puncta lachrymalia and canaliculi have been observed as congenital anomalies, and Majewski has observed quadruple puncta. There may be congenital absence of these structures, or the lacrimal points may be wanting and the canals may be represented by furrows along the edge of the lid.

2. **Acquired Anomalies.**—The slightest change in the natural relation of the lower punctum to the eye, against which it is directed backward, causes *epiphora*, or an overflow of tears.

The most fruitful sources of such abnormal relationship are the various chronic inflammations of the lid and conjunctiva—blepharitis, granular conjunctivitis, and ectropion—and facial palsy and wounds of this region. In facial palsy, watering of the eye is sometimes an early symptom, and is caused partly by the loss of the compressing power of the lid, especially in the fibers of Horner's muscle, and partly by the falling away of the punctum. An overflow of tears may follow an abnormal position or enlargement of the caruncle. All these conditions cause a *malposition* of the *punctum lachrymale*.

Epiphora is also caused by a stye or tumor of the lid near the punctum, or, if the canaliculus is closed, by the presence of a foreign body, usually a cilium; by a mass of fungus (*streptothrix*), which, by becoming calcified, may form a so-called *tear-stone*, and by a *polyp*. In like manner chronic conjunctivitis and marginal blepharitis may close either the lacrimal point or the canaliculus. These affections are included under the terms *stenosis* of the *punctum lachrymale* and *obstruction* of the *canaliculus* (for additional causes of epiphora, see page 584).

Treatment.—If a foreign body is present, it is usually necessary to slit the canaliculus in order to remove it.

In many cases of epiphora which depend simply upon closure of the lacrimal point this may be opened by means of a gold or silver pin or a *dilator*, which is pushed along the canaliculus. Afterward the permeability of the lacrimal duct may be tested by inserting the point of an Anel syringe and injecting boric acid solution and observing whether it passes freely into the nose. This very simple procedure will often afford great relief without the necessity of either slitting the canaliculus or dilating the duct. If the epiphora has been caused by facial palsy, the treatment advised does not apply.

Anomalies of the Lacrimal Sac and Nasal Duct.—1. **Dacryocystitis.**—The symptom in affections of the lacrimal sac and nasal duct which is always present is epiphora; the eye swims in tears, and these are excited to overflow by exposure to dust, cold, or wind; the caruncle and plica are swollen; the neighboring conjunctiva is hyperemic and injected (*lacrimal conjunctivitis*); the skin is macerated, and the margins of the lid, especially toward the nose, show signs of blepharitis.

Usually there is slight distention over the region of the lacrimal sac (*mucocele, lacrimal tumor*), and pressure upon this expresses through the puncta the retained fluid, which is a clear or semitransparent viscid mucus (*dacryocystitis catarrhalis*), or turbid from mixture with purulent material (*dacryocystitis blennorrhoica*).

This *chronic* distention of the lacrimal sac is liable to develop into a suppurative inflammation producing *acute dacryocystitis,* which may be preceded by fever and chill; the lids and region of the nose become tense and tender to the touch, and a red and brawny swelling resembling erysipelas, for which it not infrequently has been mistaken, overspreads the region.

Should a phlegmonous inflammation involve the cellular tissue (*dacryocystitis phlegmonosa*) which surrounds it, the pus burrows in front of the sac, forms pouches in the connective tissue, and in most instances the *lacrimal abscess* thus formed points below the *tendo oculi.* If unmolested, the abscess ruptures externally with the formation of a fistulous opening into the sac, the mouth of which is surrounded by pouting granulations (Fig. 242).

Fig. 242.—Phlegmonous dacryocystitis; pouting granulations surround the fistulous orifice (from a patient in the Children's Hospital).

2. **Prelacrimal Sac Abscess.**—This consists of a swelling above the internal palpebral ligament and a little external to the region of the lacrimal sac, associated with a fistulous opening, from which pus flows, having no connection with the sac itself. It may be caused by a blow at the inner angle of the eye and may be associated with caries and perforation of the lacrimal bone (Bull). The same condition appears without injury in children who are the subjects of hereditary syphilis.

The condition is to be distinguished from a true lacrimal abscess by the fact that there is no interference with the passage of tears from the conjunctiva into the sac, and by the absence of acute inflammation. *Prelacrimal sac cysts* are described, and small tumors may appear in this region. One removed and examined by the author had all the histologic appearances of tubercle.

The *treatment* is that of an abscess, together with such constitutional measures as may be indicated by the dyscrasia of which the patient is the subject.

3. **Fistula of the Lacrimal Sac.**—This occasionally has been observed as a congenital anomaly, and may be present on only one side or on both sides. The opening is usually directly under the internal palpebral ligament.

Generally a fistulous opening into the sac is caused by the *rupture of a lacrimal abscess,* but it may result from a carious condition of the

upper canine teeth. The opening may appear about 1 cm. below the punctum, but also in various spots along a line which runs outward, parallel to the lower orbital border.

It usually communicates with the sac, but in rare instances the opening may lead into the lower canal only, the sac above being shrunken. Pus and mucopus, and later tears, which should descend into the duct, exude from the opening, which for a long time persists as a fine orifice, at the mouth of which appears a drop of clear fluid. This is the so-called *capillary fistula.*

The condition is to be differentiated from a *buccal fistula* below the margin of the orbit, by observing that in the latter the situation is never accurately at the orbital margin, that a sound never passes upward, but only downward, laterally, or posteriorly, and that the secretion is always purulent.

4. **Obstruction of the Nasal Duct.**—This generally antedates the affection of the sac. It may be situated at any part, but selects by preference the point at which the nasal duct enters into the sac, or the lower end where it passes into the nasal chamber.

In the early stages of catarrhal dacryocystitis there probably is no true stricture of the duct, but the flow from the sac into the nose is prevented by swelling of the mucous tissue; later, and in other instances, dense cicatricial strictures occur.

Causes of Disease of the Lacrimal Sac and Nasal Duct.—Disease of the lacrimal sac is rarely primary. In young infants dacryocystitis, often double, is not infrequently seen—*lacrimal blennorrhea* or *lacrimal abscess of infants.* Donald Gunn thinks that the cause of mucocele of newborn children, becoming afterward dacryocystitis, depends upon a dilated duct, the dilatation being brought about during fetal life by obstruction at the lower end, depending, for example, upon some developmental fault. The pus usually contains pneumococci; staphylococci, streptococci, and Bacterium coli are also occasionally present. Rochon-Duvignaud has shown microscopically that there may be an occasional congenital obstruction at the lower end of the nasal duct.

Both a local and a general disposition to tear-duct troubles has been assumed by some authors, and by others, for example, Haab, hereditary predisposition has been given etiologic prominence. The female sex suffers more frequently than the male, and the left tear-duct is more often diseased than the right (Cahn).

In the majority of cases blennorrhea of the sac is caused by a retention of the secretion on account of stricture or obstruction in the nasal duct, and the participation of the lining of the sac in an inflammation of the nasopharynx. In other instances strictures result from, rather than cause, the blennorrhea. A proper appreciation of the pathologic conditions of the nasal mucous membrane in relation to diseases of the lacrimal apparatus is of the utmost importance, and in nearly every case of disease of the lacrimal sac and of the lacrimonasal duct morbid conditions of the nasal chambers and of the nasopharynx are present, especially tumefaction of the mucous membrane, hypertrophy, and

abnormal position of the turbinate bones, strictures after nasal ulcers, and caries of the nasal bones. Of great importance in this regard, especially in suppurative dacryocystitis, is infection of the ethmoid cells and of the antrum of Highmore, and in a search for a primary cause these regions deserve accurate investigation.

Although it might seem natural that conjunctivitis, and especially purulent conjunctivitis, should cause lacrimal disease, this is by no means frequently the case. Conjunctivitis and blepharitis, so often accompanying disorders, follow rather than cause the lacrimal affection.

Obstruction of the duct and disease of the sac are sequels of measles, scarlet fever, and especially small-pox, because these exanthems are accompanied by inflammation of the nasal mucous membrane.

Periostitis and caries of the lacrimal bone, the result of syphilis, are important causes. Gummy growths may block the sac and go on to rapid suppuration. Igersheimer calls especial attention to the frequency with which hereditary lues is a cause of disease of the lacrimal passages in children. *Tuberculosis of the lacrimal sac* is of not infrequent occurrence.

The relation between asymmetry of the face and disease of the lacrimonasal duct deserves mention; indeed, Hasner assumed that a local disposition to these disorders depended upon this asymmetry. Traumatism accounts for certain cases. Most impermeable obstructions follow injuries and the rough use of bougies. Stoppage of the lacrimonasal duct may be caused by pressure from neighboring tumors—for example, in the antrum of Highmore, and by foreign bodies lodged in the lower lacrimal canal and in the nasal chambers. *Actinomycosis* of this region has been reported (von Schroeder).

Fistulas, especially those seen in infants, often arise from disease of the bone, which, in turn, is the result of inherited syphilis.

Tumors of the lacrimal sac are uncommon; epithelioma (Pasetti), sarcoma (T. H. Butler), and plasmoma (Verhoeff and Derby) have been observed.

Prognosis in Lacrimal Disease.—The well-known fact that under the most skilful treatment affections of the tear-passages often resist healing renders a guarded prognosis necessary. This depends entirely upon the condition of the nasal chambers, the duration of the malady, the permeability of the stricture, and the cause of the trouble. If the latter is the result of injury the prognosis becomes especially grave, and the malady may be irremediable.

Character of the Lacrimal Secretion under Pathologic Conditions.—The lacrimal sac is a reservoir for the fluid secreted by the conjunctiva, and this fluid is more or less loaded with micro-organisms. The Streptococcus pyogenes, pneumococcus, and other pathogenic organisms are always present in dacryocystitis. If the cornea is abraded, or if a solution of continuity in this membrane is necessitated by an operation, the presence of these organisms in the fluid becomes a serious complication. They may turn a simple abrasion into a sloughing ulcer or an aggravated hypopyon-keratitis. They may prevent the healing of

an ordinary keratitis, and finally they may inoculate an operative wound and defeat the object of the operation. For this reason it is most important that in any of the three conditions just quoted the permeability of the nasal duct should be ascertained. If it is strictured, it should be opened, and the walls of the lacrimal sac, if inflamed, brought to a healthy condition as speedily as possible, or the sac should be extirpated. The importance of this relation of the lacrimal apparatus to diseases of the cornea and to the prognosis of cataract operations has been elsewhere described.

Treatment of Diseases of the Lacrimal Sac and Duct.—Conservative measures should always be tried first—viz., intranasal treatment, massage over the sac while the inner canthus is kept filled with an antiseptic liquid, and dilatation of the punctum and irrigation of the sac. Many cases of simple epiphora are due to ametropia and heterophoria and even to various nervous diseases—for example, tabes dorsalis and neurasthenia—hence operative interference is to be deprecated unless the exact cause of the condition is ascertained. Epiphora may be an early sign of exophthalmic goiter (Berger, J. T. Carpenter).

In organic cases, usually, the following procedures are recommended: slitting the canaliculus, introducing a probe into the nasal duct, and syringing the sac and nasolacrimal duct; or, in the presence of proper indications, excision of the lacrimal sac. The method of slitting the canaliculus and the introduction of a probe are described on page 681.

After the canaliculus has been dilated or incised, the duct and the sac should be washed out thoroughly with some antiseptic fluid—a saturated solution of boric acid or a 1 : 5000 solution of bichlorid of mercury, or formaldehyd 1 : 3000, or permanganate of potassium 1 : 5000, or protargol or argyrol, 5 to 10 per cent. Argonin, 2 to 4 per cent., has been recommended. Great care should be employed in using solutions of argyrol and protargol, lest they escape into the surrounding structures and produce unsightly staining of the skin.

Some surgeons, as a rule, split the upper canaliculus, although the usual practice is to approach by means of the lower passage. If there is much distention of the sac, it has been suggested to enter the upper passage and incise both this and the wall of the sac.

In making use of probes, it is advisable to begin the first trial with a No. 1 conical probe (Bowman's or Williams'); if this fails, a smaller one may be tried. Either rapid or gradual dilatation is employed, the latter being the preferable method. Undue efforts should never be used, as it is extremely easy to make a false passage and perforate the delicate structure of the lacrimal bone, while roughness in the use of probes, by scraping off the mucous membrane, may cause the most impermeable type of stricture. Often it is not necessary to use probes at all, but simply by means of a syringe introduced into the mouth of the sac to medicate the inflamed tissue, which, as before stated, sometimes causes occlusion by swelling, without the actual presence of a cicatricial stricture.

Sounds should be used at first every second or third day, but as the case progresses longer intervals may elapse. Large probes (4 mm. in diameter) are advocated by Theobald and Snell, but, in the author's experience, are not only unnecessary, but often harmful.

If a lacrimal abscess supervenes and is seen early, the canaliculus should at once be slit and, if possible, the secretion evacuated and the passage into the nose restored. Frequently the pain and swelling are such as to render this impossible, and the opening must be made upon the face, about 1 cm. below the palpebral tendon, cutting downward and outward. The cavity should then be thoroughly cureted, packed with gauze, and allowed to heal gradually from the bottom.

An excellent practice is to use hot compresses over the swelling, preferably of carbolized water, at a temperature of 120° F., frequently changed and applied for five or ten minutes at a time. Later the passage into the nose may be rendered patulous with probes, in the manner already described. The practice of introducing a lead or silver style the author has abandoned, although many surgeons are strongly in favor of its use, especially the use of a lead style, which is preferable to a canula (H. Moulton). The passage of bougies of gelatin impregnated with 30 to 50 per cent. of protargol has been recommended (Antonelli).

The treatment of dacryocystitis of infants should consist in the use of a simple collyrium, boric acid or boric acid and sulphate of zinc, frequent evacuation by pressure of the contents of the sac, and gentle massage. In the experience of the author this is usually sufficient; rarely slitting the canaliculus and passing probes may be necessary.

Swelling over and around the lacrimal sac, together with fistulous communication into it, occasionally will subside under the judicious use of a compressing bandage.

The great difficulty that is sometimes experienced in keeping the canaliculus open has led to the employment of *electrolysis*. This is applied to the canaliculus by means of a probe fitted in a handle and connected with the negative pole of a battery, the positive electrode being placed on the back of the neck. The *séance* should last about half a minute with a current of 2 milliamperes.

In addition to the local measures already mentioned for the purpose of producing healing in cases of lacrimal disease associated with a catarrhal condition of the passages, solutions of nitrate of silver, salicylic acid, iodoform, aristol, creolin (1 per cent.), and blue pyoktanin (1 : 1000) have been advocated.

In acute inflammation with abscess formation, quinin and iron in the form of Basham's mixture are indicated; in syphilis, with disease of the bone and gummy deposit, the usual drugs should be exhibited; in struma, cod-liver oil, hypophosphites, and iron, in the form of the syrup of the iodid, are the most trustworthy remedies.

Scrupulous attention to the nose and the nasopharynx is necessary, and any local lesions which present themselves must be treated. In the absence of a special line of practice for this region excellent results

follow a simple spraying of the parts with Dobell's solution or peroxid of hydrogen one-third, water two-thirds, while carrying on the regulation measures for the relief of the lacrimal disorder. If there is decided disease of the region, the proper treatment of the part with the view to removing diseased structures should be undertaken. The importance of examining accessory nasal sinuses has been pointed out, especially the ethmoid and the maxillary sinus.

Occasionally it will happen that although a duct has been thoroughly opened, the probe passes readily, and the liquid used in the syringe flows freely from the nose, the epiphora continues, and the eye fairly swims in tears. In such circumstances a probe should be passed into the nose and the entrance of the duct into the inferior meatus properly exposed by means of a nasal speculum. Quite often it will be seen that a thickening of the duct entrance, or perhaps a valve-like flap of mucous membrane, occludes the passage. This is pushed aside by the probe or forced aside by the liquid when it is injected, but entirely stops the flow of the tears. This simple precaution will sometimes lead to the discovery of the cause of failure to relieve cases which have stubbornly resisted treatment.

If a fistula remains, this may sometimes be closed, as already stated, by compression. In the event of failure, freshening of the edges and the galvanocautery may be tried, the surrounding pouting granulations being removed by scraping. The capillary fistulas are productive of no inconvenience and may be allowed to remain undisturbed.

Extirpation of the lacrimal sac is indicated, and usually yields good results in many cases of chronic dacryocystitis. It may be employed if conservative and ordinary surgical measures have failed, if the patient cannot or will not devote sufficient time to treatment, if there is an impassible stricture, if an operation on the eyeball is speedily necessary, if there is a serpiginous ulcer of the cornea (Kuhnt), and in cases of caries of the lacrimal bone. The operation is further indicated in those whose occupation exposes them to corneal injury (Axenfeld) and in insane patients. The operation is so satisfactory in its results that, in the author's opinion, it should in large measure replace the use of probes and the other measures which have been described. If, subsequently, the epiphora is annoying, *extirpation of the lacrimal gland* has been performed, and is especially advocated by C. R. Holmes. In place of complete excision, removal of the palpebral gland may be tried. (For methods of operating, see page 684.) Usually, however, as the conjunctiva resumes its normal condition, the epiphora, under ordinary conditions at least, ceases to be annoying, and often disappears. (For other methods of operating in cases of dacryocystitis, see page 682.)

CHAPTER XXI

DISEASES OF THE ORBIT

Congenital Anomalies.—*Anophthalmos*, or complete absence of one or both eyes, is an affection which, like the other congenital anomalies, more frequently is double than one sided. A child born without eyes may be healthy and well developed in other respects, or may be the subject of additional congenital deformities. The palpebral fissures are small, the lids usually deficient in size, sunken, and upon their separation the empty orbit without trace of the globe is revealed. Sometimes, however, careful dissection will expose a rudimentary eyeball at the apex of the orbit.

The most reasonable explanation of this anomaly is that no primary optic vesicle has budded out from the anterior primary encephalic vesicle, or that, having budded out, it has failed to form a secondary optic vesicle.

Microphthalmos and *megalophthalmos* are anomalies of the globe to which reference has been made. Sometimes *cysts* of bluish hue are connected with rudimentary eyes, the cyst being evident in the lower part of the orbit or the lower lid—*orbitopalpebral cyst.* Zentmayer and Goldberg have found retinal elements in a cyst of this character.

Cyclopia is a congenital malformation characterized by a fusion of the orbits and the two eyes in the middle of the face, so that there is only one eye situated in the place normally occupied by the root of the nose.

General Symptoms of Orbital Disease.—Two symptoms are so constantly present that they may be said to be essential to the clinical picture of most of the affections of the orbit:

1. *Proptosis or Exophthalmos.*—This consists of more or less protrusion and displacement of the globe.

2. *Immobility of the Eyeball.*—This may be complete or partial, and, if vision is unaffected, the limitation of the movements of the eye is associated with diplopia. Complete immobility may be differentiated from a similar condition due to palsy of all exterior ocular muscles (ophthalmoplegia externa) by the absence of ptosis (Noyes).

The following signs may also be associated with orbital disease:

(*a*) *Chemosis of the conjunctiva*, either universal or else localized upon a special portion of the globe, indicating the neighborhood of the diseased area.

(*b*) *Redness, swelling*, and *edema* of the eyelids, especially in inflammatory affections of the cellular tissue of the orbit and disease of the accessory nasal sinuses.

(*c*) *Pain*, most noticeable when the patient attempts to move the eye or when the surgeon palpates the globe and presses it inward. In addition to the pain in the orbit itself, *frontal headache* is a common symptom, especially if the sinuses are involved, and *tenderness on pressure* along the margin of the orbit and accessible portions of its walls is one sign of disease of the periosteum.

(*d*) *Fluctuation* occurs, but not constantly, if an abscess of the orbit has formed.

(*e*) *Disturbance of Vision.*—In some cases of orbital diseases there is no disturbance of vision; in others there may be marked changes in the eye-ground—papillitis (choked disk), atrophy, hemorrhages, and vasculitis or perivasculitis.

Periostitis of the orbit is both *acute* and *chronic*, and in the acute type appears either as a *localized* affection or as a *diffuse* suppurative process.

The **symptoms** of acute localized periostitis are pain, tenderness over the seat of the disease, usually the margin of the orbit, injection and chemosis of the conjunctiva, and some swelling of the lids and protrusion of the ball. In the diffuse variety of the disease all the foregoing symptoms are much aggravated, and there may be, in addition, fever, general headache, delirium, and stupor. In such a case the differential diagnosis between it and an orbital cellulitis becomes extremely difficult. In fact, the cellular tissue is associated with the periosteum in the inflammation. A *subperiosteal abscess* may form and become encapsulated or, passing forward, burst through the skin of the eyelid or at the angles of the orbit and form an *orbital fistula.*

In chronic periostitis there are deep-seated pain, often worse at night, tenderness on pressing the eyeball backward, thickening of the tissue beneath the orbital margin, and swelling of the lids and conjunctiva, although the latter symptoms, together with proptosis, may be absent.

According to Mraček, syphilitic periostitis most frequently attacks the orbital margins, and may occur in a *gummatous* or a *sclerosing* form. It less commonly involves the orbital walls behind Tenon's capsule, and is then generally gummatous in type. The site is usually in the upper or outer wall, and the disease causes trigeminal neuralgia, worse at night, and restriction in the mobility of the globe, with squint and diplopia. Optic neuritis may occur.

The **causes** of periostitis, especially of the chronic form, in addition to syphilis, in which disease it is sometimes a secondary, but more often a late, manifestation, are rheumatism, tuberculosis, injuries and affections of the sinuses, notably the frontal and the ethmoid.

The **prognosis** depends upon the type of the disease. If localized, this is favorable; if diffuse and suppurative, not only may extensive implication of the tissues surrounding the globe leave permanent disabilities and deformities (exophthalmos, muscle palsy, optic-nerve atrophy, necrosis), but the inflammation may extend to the meninges of the brain and cause death.

Chronic periostitis may last for months, and in any type fistulas, necrosis, and caries of the bone are the common result. Periostitis due to syphilis presents the most favorable prognosis.

Treatment.—The constitutional treatment depends upon the cause, and includes the iodids and salicylates in rheumatic cases, and the use of mercurials and salvarsan in syphilitic cases.

The surgical treatment of acute periostitis consists in an incision into the affected area and evacuation of the pus; in short, the treatment is the same as that applied to acute periosteal disease elsewhere located. The relation of periostitis to sinus disease demands a careful examination of the sinuses and treatment according to the findings.

Caries and Necrosis.—Caries is prone to attack the margin of the orbit, especially the lower and outer part, and may be due to syphilis or tuberculosis. An injury often is the exciting cause.

The **symptoms** of periostitis are present, suppuration develops, the abscess comes to the surface through the lid over the diseased area, rupture occurs, with the discharge of pus, a fistula forms, surrounded by granulations, and through this a probe will detect the softened bone. Very decided deformity of the lid may be occasioned, most commonly in the form of an ectropion (compare Figs. 95 and 98).

Caries of the orbit is most common in children, and, as has been pointed out, selects the margin of the orbit for its site, although it may occur in the roof, in which case it becomes a complication endangering life, owing to the proximity of the brain. The inflammation may spread to the orbital tissues and cause exophthalmos and neuroretinitis.

Necrosis of the orbit is much less common, and its immediate cause is an osteitis occurring as a consequence of acute periostitis. A fragment of bone completely separated by a fracture from the periosteal surroundings would probably undergo necrosis, and the rough use of probes may cause mortification of the delicate lacrimal bone. Necrosis, unlike caries, is more common in adults.

Treatment.—This consists of the remedies recommended in the treatment of periostitis, and, as caries is a very chronic affection and most common in strumous (tuberculous) subjects, cod-liver oil, phosphates, and iodid of iron should be included in the constitutional measures, and should be exhibited for long periods of time.

The local treatment during the early ulcerative stage of caries consists of evacuation of foci of suppuration, careful cleansing with antiseptic solutions, and drainage. Considerable caution is necessary before resorting to the removal of the diseased bone with a gouge, because the process is essentially chronic and may be aggravated by the manipulations of the instrument; but roughened bone should be scraped with a sharp spoon and the diseased portions thoroughly removed. If the roof of the orbit is affected, great care is necessary lest the cranial cavity be penetrated. If a piece of the orbital wall has undergone necrosis, this should be removed when it has become loose or detached. The regions overlying the sinuses should be carefully examined.

Cellulitis (*Phlegmon of the Orbit*).—There are several varieties of inflammation of the cellulofatty tissue of the orbit. Thus the inflammation may be acute, subacute, or chronic, unilateral or bilateral, and finally it may undergo resolution or, as more commonly is the case, terminate in suppuration.

In the *mild* form the *symptoms* are dull pain, swelling of the lids, slight exophthalmos and diplopia, without inflammatory symptoms and without constitutional disturbance.

In the *acute* phlegmonous variety of the disease there are chills, fever, deep-seated pain, most marked upon attempting to move the eyes, general headache, exophthalmos, limitation in the movements of the eye (which may become entirely fixed), and swelling and edema of the lids, together with hyperemia and chemosis of the conjunctiva. The last two symptoms are so severe at times as to give at first sight the general impression of a violent attack of purulent conjunctivitis (Fig. 243).

Fig. 243.—From a photograph of a patient in the Philadelphia General Hospital suffering from double orbital cellulitis the result of erysipelas.

In the earlier stages vision is not usually affected, but later there may be optic neuritis followed by atrophy, dilatation of the pupil, anesthesia, and even ulceration of the cornea, and, indeed, in severe cases the eyeball may suppurate. In certain types of orbital cellulitis extensive intra-ocular changes occur, with hemorrhages and vascular alterations, due to compression of the central vessels of the retina producing stoppage of the circulation and edema and exudation into the retina (Knapp). Blindness from orbital abscess may be due to retrobulbar necrosis of the optic nerve, from thrombosis of the pial vessels, and of the central vessels (Bartels). Fluctuation finally develops, and pointing usually occurs below the inner portion of the supra-orbital ridge.

The symptoms of *chronic* abscess are much less violent and distinctive than those just described. They may, indeed, be mistaken for other morbid conditions, especially as the abscess is commonly associated with diseased bone or periosteum in scrofulous subjects, or may occur in them from an injury or the presence of a foreign body.

The **causes** of orbital cellulitis are various. It may be traumatic or may be due to exposure to cold; it may follow in the wake of scarlatina, typhoid fever, or influenza; or it may be the result of a meningitis. The most violent types of orbital cellulitis occur with facial erysipelas. In these instances the affection is usually double. The extension of inflammation from diseased teeth or suppuration in the ethmoid cells, sphenoid, or antrum of Highmore may cause the

affection. Birch-Hirschfeld's investigation demonstrates that the largest number of orbital inflammations (about 60 per cent.) are due to accessory sinus inflammation. The infection is conveyed from the sinus to the orbital contents by means of septic thrombosis, thrombophlebitis, lymphangitis, or erosion of the bony partition. Finally, a certain number of cases are metastatic, and develop in the course of pyemia, especially puerperal septicemia. The association of orbital cellulitis with periostitis has already been referred to, and a certain amount of cellulitis occurs whenever there is a general inflammation of the globe.

Progress and Prognosis.—In mild cases the prognosis is favorable; in severe cases, unfavorable; and in double cases, especially those which have originated under the influence of erysipelas, usually fatal. Although the pus may make its exit through the conjunctiva or eyelid, it may also pass backward through the sphenoid fissure. In pyemic cases, and, indeed, in the course of any severe inflammation of the cellulofatty tissue of the orbit, *phlebitis of the orbital veins* may become a complication and extend to the cavernous sinus, leading to a fatal termination. If the disease passes to the cavernous sinus upon the opposite side, the other eye also becomes involved and exophthalmos is evident.

In framing a prognosis it is necessary to consider the effect of the disease upon the eyesight and upon the life of the patient. Sight may be impaired or destroyed by the development of optic neuritis, optic-nerve atrophy, exudation and hemorrhages into the retina, or by suppuration of the cornea; life may be endangered by an extension of the suppurative process into the cranial cavity, or by the original malady which caused the cellulitis.

Treatment.—The general treatment should include supporting measures and iron and quinin. Occasionally the pus points in the conjunctival sac and may be evacuated by an incision through the conjunctiva between the ball and the side of the orbit, care being taken not to injure the ocular muscles and to secure good drainage afterward. In deeper situations the purulent focus is best reached by a curved incision made over the orbital ridge which divides the periosteum, which is next separated with an ordinary bone elevator, kept well between the bone and the periosteum, thus avoiding the levator, the tendon of the superior oblique, and the lacrimal gland. If pus does not immediately present, the depth of the orbit must be explored with a probe until the pocket of pus is found, and evacuated by an incision through the periosteum. Drainage may be secured with iodoform gauze or an ordinary drainage-tube. The position of the original incision is determined by the probable situation of the pus; that is to say, whether it is made along the upper, lower, inner, or outer orbital margin. If the source of the pus is from the ethmoid, the orbital plate of this bone should be perforated, carious bone and necrotic tissue removed, and a drainage-tube carried from the orbit, through the ethmoid, into the nose, which not only secures an adequate drainage, but permits the subsequent

washing out of the tract. Indeed, the frequent association of sinus disease with orbital cellulitis usually demands that the incision shall be so placed and sufficiently broad to render exploration of the orbital walls practicable and treatment of the affected sinus (frontal or ethmoid) possible.

Inflammation of the Oculo-orbital Fascia (*Tenonitis*).—This affection is characterized by swelling of the upper lid, the discoloration of the tumefied lid being limited to its tarsal portion (Pincus); pain on the slightest movement of the eye, some proptosis and limitation of movement, together with the appearance of a watery nodule or vesicle situated over one of the recti muscles; in other cases the chemosis may be more general. Primary serous tenonitis is a rare disease, about 40 cases being on record (Birch-Hirschfeld). Tenonitis may follow an injury or an operation—for instance, tenotomy; in some instances it is due to rheumatism, possibly to tuberculosis, and it has been noted as a sequel of diphtheria, typhoid fever, and epidemic influenza, and may be caused by syphilis. Lesions almost exactly analogous to those of tenonitis are occasionally caused by an intra-ocular growth; for example, a sarcoma (see page 362).

The treatment should consist of warm fomentations and, according to the indications, iodid of potassium or the salicylates.

Thrombosis of the Cavernous Sinus.—During phlegmonous inflammation of the orbit there may be thrombosis of the orbital veins, and extension from them to the cavernous sinus or to the other sinuses of the brain. Primary traumatic non-infective thrombosis of the cavernous sinus has been described by H. Knapp. Septic thrombosis of the cavernous sinus may arise as the result of any infected lesion in the area drained by the ophthalmic vein or its branches, for example, pustules on the face, nostrils, or eyelids, and from purulent affections of the accessory sinuses and rhinopharynx, and from erysipelas and wounds. According to St. Clair Thomson, next to disease of the sphenoidal sinus, pyogenic infection from the ear is the most common cause of thrombosis of the cavernous sinus. The ocular symptoms which accompany cavernous sinus thrombosis are: proptosis, edema of the eyelids and chemosis of the conjunctiva, haziness, anesthesia of the cornea, and partial or complete ophthalmoplegia—that is, gradual involvement of the third, fourth, and sixth nerves, venous engorgement of the retinal veins, and neuroretinitis. The general symptoms include headache, fever, delirium, coma, and convulsions. As the prognosis is extremely unfavorable, an operation designed to expose the sinus and remove the thrombus, the feasibility of which has been demonstrated (Hartley, Knapp), would seem to be proper.

Tumors and Cysts of the Orbit.—Tumors have been divided by systematic writers into those which originate in the orbit, but are unconnected with the globe of the eye; those which arise from the periosteum or bony walls of the orbit; those which commence in the cavities close to the orbit; and those which originate in some vascular disease within the cavity of the orbit or the neighboring por-

tions of the cranial cavity, and which usually give rise to *pulsating exophthalmos.*

Two classes of tumors, namely, those which arise from the optic nerve and those which arise from the lacrimal gland, are sometimes included among the orbital growths. They have already been discussed in another section.

The *nature* of orbital tumors is either benign or malignant, and they may be congenital or acquired, primary or metastatic.

Symptoms.—These vary according to the position, size, and density of the tumor, but in general terms are those which have been narrated as more or less common to all diseases of the orbit. With regard to the protrusion it may be said that a tumor within the cone of the recti muscles is apt to cause a forward displacement of the globe, while one situated outside of this cone may displace the eyeball in some particular direction.

Considerable proptosis may occur under the influence of an orbital tumor without causing the globe to protrude between the fissure of the lids. This is due to the fact that the lids are extensible and accommodate themselves to the increasing volume behind them; finally, however, the protrusion may be so great that the lids can no longer close over the prominent ball.

Prognosis.—This depends upon the nature of the tumor, the density of its tissue, the rate of its growth, and the availability of surgical interference.

Treatment.—In dealing with benign tumors and some encapsulated sarcomas the eyeball, if uninvolved, should be allowed to remain, if possible; but if the tumor is malignant the eye should be excised, in most instances, with the entire contents of the orbit (see chapter on Operations). According to C. S. Bull, encapsulated tumors of the orbit may be removed with the almost certain hope of favorable result, while non-encapsulated tumors present a most unfavorable prognosis. After removal or in the event of a return, the *x*-rays should be employed. Recently radium has been used with success in the treatment of orbital growths. As was pointed out by Panas, Snell, and others, certain apparently organic tumors of the orbit, probably lymphomas, occasionally disappear under medicinal treatment—for example, iodid of potassium, arsenic, etc. Hence the necessity of careful medication before surgical measures are tried. The difficulty of distinguishing an orbital tumor from a chronic inflammatory process is often great and no method of diagnosis should be omitted, especially careful examination of the accessory sinuses. In this regard *x*-ray examination is important.

1. **Tumors which Originate in the Tissues of the Orbit.**—These include cysts, fibromas, cavernous and simple angiomas, lymphangiomas, lipomas, enchondromas, lymphomas, cylindromas, endotheliomas, and various types of sarcoma. Discrete lymphoid infiltration of the orbit, causing proptosis, has been reported by Coats. Carcinoma, except in connection with the lacrimal gland, does not occur in this situation

as a primary tumor; it may, arising from the lids or conjunctiva, grow inward and involve the orbit. In a total of 68 cases of metastatic carcinoma of the choroid there was in 13 of them an extra-ocular extension of the growth from the focus in the choroid (Shumway). Metastasis of carcinoma from distant organs to the ocular muscles has been described (see page 535). Adrenal tumors in children, usually under four years of age, with metastasis to the orbit, have been reported (Quakenboss, Verhoeff).

Sarcomas of the orbit may be primary or metastatic and may present the various types of cellular structure characteristic of these tumors. Some sarcomas of the orbit should be classified with the *endotheliomas*. If the morbid process is an extensive one, radical removal of the entire contents of the orbit is the only procedure, and subsequently the *x*-rays or, preferably, radium should be employed. Even in inoperable malignant disease of the orbit pain is lessened by the application of the

Fig. 244.—Metastatic sarcoma of the orbit (from a patient under the care of Dr. Wharton in the Children's Hospital).

Fig. 245.—Sarcoma of the orbit springing from the periosteum over the great wing of the sphenoid.

x-rays, and the complete disappearance of sarcoma of the orbit, without operation, under the influence of repeated applications of Röntgen rays has been reported (L. W. Fox), and represents a therapeutic measure deserving of the most thorough trial. In like circumstances radium may be used. Encapsulated sarcomas may occasionally be removed with preservation of the eyeball. *Traumatic sarcomas* offer a most unfavorable prognosis, and operation hastens rather than retards the fatal issue. Sarcomas of the orbit should not be confounded with those which arise within the eyeball and have burst their boundaries (see page 362).

Berlin divides *orbital cysts* into two principal groups, *cephaloceles* and *true cysts*. Cephaloceles are located at the root of the nose, and extend to the brow, nasal cavities, or orbit. Characteristic of cephaloceles and meningoceles is the fact that they present at the inner side of the orbit, that they fluctuate and are transparent. True cysts should be divided,

according to Klingelhoffer, into (*a*) true cysts from constriction, which are derived from congenital meningoceles; (*b*) extravasation-cysts—that is to say, blood-cysts, hematomas, etc.; (*c*) exudation-cysts, which are very rare; (*d*) dermoids, which are the most frequent cystic tumors growing in the orbit; (*e*) mucous cysts, which may communicate with the nose, and (*f*) echinococcus cysts. Extravasated blood in the retrobulbar tissue may become encapsulated and simulate a blood-cyst, and subperiosteal blood-cysts have been described (Denig, Lamb).

Occasionally a simple incision suffices to cure a cyst if the cavity is afterward frequently syringed with an astringent or antiseptic lotion. After the evacuation of a dermoid cyst Buller recommended the introduction of a crystal of nitrate of silver or tincture of iodin to destroy the cyst wall. Usually the cyst wall must be dissected out, and, if semisolid or solid contents are present, entire removal is necessary. Care must be taken not to confound a cephalocele with an orbital cyst.

2. **Tumors which Arise from the Periosteum or Bony Walls of the Orbit.**—These include:

(*a*) *Sarcomas,* which arise from the periosteum.

(*b*) *Thickening of the periosteum*, which may simulate a true tumor, especially if the underlying bone is hypertrophied (hyperostoses: these may be multiple or diffuse), and—

(*c*) *Exostoses.*—The latter are very hard tumors having an ivory-like shell and a nucleus of spongy bone, their anatomic structure in general being like that of the osteomas proceeding from adjacent cavities.[1] All orbital osteomas grow slowly—the external exostoses more slowly than the bony tumors which originate from the frontal and ethmoid sinuses. They spring from the periosteum, and are generally found at the upper border of the orbit, although they may occur at any portion of the orbital border, and are recognized by their dense hardness and evident connection with the bone.

They may arise from injury; sometimes they are congenital, and often their origin is obscure.

The operation for the removal of an exostosis, after its exposure by suitable incisions of the soft parts and periosteal covering, consists in drilling it away at the base and completing the separation by means of a hammer and chisel. An electric drill and saw is of special service in these operations.

3. **Tumors which Arise in Cavities or Tissues Close to the Orbit.**—These include:

(*a*) *Encephalocele*, a rare condition, which appears in the form of a somewhat pulsating, fluctuating protrusion at the inner angle of the orbit; it is of congenital origin (see also page 580).

(*b*) *Nevi, epithelioma*, and *lupus*, which may extend from the skin of the face into the orbit.

(*c*) *Polypi* from the nasal chambers and surrounding sinuses, and—

(*d*) *Osteomas* of the frontal and ethmoid sinuses.

[1] For a valuable paper by J. A. Andrews, on "Osteomas of Orbit," see Medical Record, September 3, 1887.

An *osteoma* consists of a dense growth, with predominance of the ivory shell, and only a trace of spongy tissue (occasionally the reverse occurs). Generally the surface is covered with a delicate connective-tissue envelope, and part of this may be the seat of polypoid growths coming from the remains of the mucous membrane which atrophies under pressure of the tumor.

According to Andrews, osteoma of the frontal sinus first makes its appearance by a tumor at the upper inner angle of the orbit, and may be associated with the formation of polypi and suppuration of the sinus. One which grows from the ethmoid sinus first appears at the inner angle of the orbit, and the eyeball is displaced laterally.

If an osteoma springs from the antrum of Highmore, the tumor appears behind the lower eyelid, and the eyeball is displaced upward; if it arises in the sphenoid fissure, sight is affected by compression of the optic nerve.

Fig. 246.—Pulsating exophthalmos (from a case under the care of Dr. Kent Wheelock, Fort Wayne, Indiana).

Extirpation of osteomas in the sinuses is attended with considerable risk, and a number of fatal cases are on record.

4. **Tumors which Originate in Some Vascular Disease within the Cavity of the Orbit or in the Neighboring Portions of the Cranial Cavity (Pulsating Exophthalmos).**—Under the name *pulsating exophthalmos* a number of conditions of diverse origin have been described, and more than 300 cases are now on record. The conspicuous symptoms which may arise in the course of this disease, although, naturally, not all of them are present in each case, are as follows: Exophthalmos, most frequently with the eye displaced outward and downward; bruit, usually heard over the eye and above the orbit, but sometimes audible over the whole skull and evident to the patient in a roaring, humming, buzzing, or hissing sound; pulsation, which may be visible or demonstrable only by palpation, or by pressing the globe backward into the orbit; distention of the veins at the inner angle of the orbit, especially enlargement of the angular vein, and of those of the lid and even of the forehead and on the surface of the conjunctiva; corneal complications, usually in the form of exposure keratitis; frequently hyperemia of the iris and rarely actual iritis; commonly hyperemia of the nerve-head, and, occasionally, optic neuritis and even choked disk; frequently marked distention of the retinal veins and scattered retinal hemorrhages; disturbances of ocular motility, sometimes so extensive as to implicate all of the exterior ocular

muscles, sometimes only one or other of them, the external rectus being the one most frequently affected where a single muscle is involved; occasional involvement of the trifacial, of the facial, and disturbances of taste, smell, and hearing.

Formerly such symptoms were regarded as evidence of true aneurysm of the ophthalmic artery, but pulsating exophthalmos may also be due to a vascular tumor or an intracranial affection. As Rivington demonstrated, the affection may be caused by an extra-orbital aneurysm of the ophthalmic artery, aneurysm of the internal carotid, or an aneurysmal varix involving the internal carotid and the cavernous sinus. The last-named lesion—arteriovenous communication—is the one most frequently responsible for these phenomena. Dilatation from obstruction of the ophthalmic vein may cause the condition, but aneurysm by anastomosis, which may involve the orbit by spreading from neighboring parts, is not accompanied by exophthalmos. Traumatism is responsible for the majority of the cases, being the essential cause in about 60 per cent.

Treatment.—This has included: (1) Ligation of the larger arteries of the neck; (2) operations upon the orbit; (3) compression of the common carotid; (4) direct compression of the venous swellings of the eyelids and the angle of the orbit; (5) gelatin injections; (6) the administration of certain drugs and rest in the recumbent posture. Of these various procedures, ligation of the common carotid and orbital operation furnish the most satisfactory results. According to the investigations of the author and Holloway in 1907, ligature of the common carotid had been performed one hundred and fifty times, with cure or improvement in 64.6 per cent., failure in 25.3 per cent., and death in 10 per cent. of the cases; in a certain number of them, about 10 of the total number, both carotids had been ligated. The orbital operations have included ligation of the superior ophthalmic vein, of the inferior ophthalmic vein, of the angular vein, and of the smaller orbital veins, and the results in almost all of the cases have been good. If ligature of a common carotid fails before a second carotid is tied, the operation of dissecting out and tying the distended superior ophthalmic vein should be performed. If there is a distinct venous swelling in the orbit, with evident distention of the angular or superior ophthalmic vein, the operation of choice should be isolation, ligature, and resection of this venous channel. It is not without danger, and the author is aware of one fatal case, death being due to extension backward of a thrombus into the brain. Compression of the common carotid, with or without the administration of iodid of potassium, has been successful in a few instances, and may be tried before radical surgical means are resorted to.

Exophthalmic Goiter (*Graves' Disease; Basedow's Disease*).—This disease, when it is perfectly developed, is characterized by three cardinal symptoms—enlargement of the thyroid gland, palpitation of the heart, and prominence of the eyeballs. As the affection should be classified with diseases of the ductless glands, the student is referred for a full consideration of the subject to treatises upon the practice of medicine.

Inasmuch, however, as one of the cardinal symptoms—prominence of the eyeballs—is a very marked one, and as there are certain changes seen especially in and around the eyes, a few words may be added. Exophthalmos varies from a mere prominence of the eyeballs, such, for instance, as is noticeable in a highly myopic globe, to a degree of protrusion so great that the eyelids are unable to close. Excessive epiphora may be present as an early symptom—*i. e.*, before exophthalmos appears (Berger). Four symptoms should be searched for:

1. *Von Graefe's sign*, which is very important in the early recognition of the disease. Normally, when the globe is turned downward, the upper lid moves in perfect accord with it; in this disease, on rolling the eyeball downward, the upper lid follows tardily, or does not move at all. The symptom is not always present, but it may be noted prior to any exophthalmos or at least when there is only a very trifling degree of this, and it persists after the protrusion of the eye has subsided.

2. *Stellwag's Sign.*—This consists of imperfect power of winking or diminished frequency in the act; thus, there may be a number of rapid winks, succeeded by a long pause in which there is no movement of the lids, or each time that nictitation occurs, it is not complete and the margins of the lids do not, as in the normal eye, come together.

3. *Dalrymple's Sign* (Cooper-Swanzy).—This consists of retraction of the upper eyelid so that there is an unnatural degree of separation between the margins of the two lids. The widening of the palpebral fissure produces the peculiar stare which is present in the subjects of exophthalmic goiter, and which has been compared to a similar appearance produced by the action of cocain.

4. *Moebius' Sign.*—This consists in an imperfect power, or in an entire absence, of convergence, and may be sought for in the usual manner (see page 74). A decided pigmentation of the skin of the eyelids is seen in some patients with exophthalmic goiter. Gifford calls attention to a symptom in exophthalmic goiter, namely, the difficulty of everting the upper eyelid, which may be present in the early stages of the malady.

Changes in the Cornea.—The exposure to which the eye is subject and also the paralysis of the nervous supply may cause drying of the epithelium of the cornea, and ulceration of so violent a type as to produce destruction of the eye. New vessels may develop in the lower part of the cornea on account of its exposure through the widened palpebral fissure. These corneal changes necessarily occur in severe types of the disease where the protrusion of the eyeballs has been considerable.

Ophthalmoscopic Changes.—These are not commonly present to any great degree except in so far as a change in the size of the retinal vessels is concerned. The arteries may be dilated and assume a caliber larger than normal and equal to that of the veins. Spontaneous arterial pulsation is frequently present (Becker). Alterations in the optic nerve and in the general fundus are not usually found, and there are no changes in the eye-grounds characteristic of the disease.

Nature of the Disease.—The cause of exophthalmic goiter is not known; but there is little doubt that the symptoms of this disease depend upon a disturbed function of the thyroid gland, whereby there is excessive internal secretion from it, or entrance into the general system of more of its active principle than is normal (Hare).

Treatment.—For the general treatment of exophthalmic goiter the student is referred to the text-books on general medicine, surgery, and neurology. Sympathectomy has been practised for the relief of exophthalmos. Partial thyroidectomy is usually performed and the operation meets with great success. If ulceration of the cornea occurs the usual treatment is applicable. To prevent exposure of the cornea, the widened palpebral fissure may be narrowed by the operation of tarsorrhaphy (see Fig. 269).

Affections or Diseases of the Sinuses.—In discussing tumors of the orbit, it was noted that growths from the frontal sinuses, the sphenoid fissure, the ethmoid cells, and the antrum may encroach upon the orbit. The limits of this book do not permit a full consideration of this subject, for which the student must turn to special treatises. In addition to the morbid growths there remain to be briefly considered:

Fig. 247.—Acute frontal and ethmoid sinusitis. Notice the edema of the lid and upper and inner portion of the orbit (from a patient in the University Hospital).

1. **Disease of the Frontal Sinus.**—This is most often a distention of the frontal sinus by mucus (*mucocele*) or pus (*empyema*). Abscess has been attributed to postnasal catarrh, syphilis, tuberculosis, and periostitis, and is due to the stoppage of the normal outlet, thus causing the accumulation of secretion until the sinus becomes filled, its walls distended and thin, and a tumor presents, usually at the upper and inner angle of the orbit. It may occur under the influence of erysipelas, acute infectious diseases, and epidemic influenza. Sensitiveness on pressure over the frontal bones and frontal headache or supra-orbital pain are common and somewhat characteristic symptoms, and are especially marked in *acute* frontal sinusitis. The protrusion may cause displacement of the eyeball downward and outward and diplopia, and the pressure upon the lacrimal sac, epiphora. Coryza and purulent discharge from the nostril may be present. According to Bull, if a dense, hard swelling appears at the upper and inner angle of the orbit, which is slow in growth and painless, an osteoma of the sinus is almost

certainly present. In rare instances the abscess in the sinus is bilateral. The *x*-rays should be used to establish a diagnosis between osteoma and mucocele of the sinus. A stereoscopic radiogram is essential in the study of accessory sinus disease, and careful *transillumination* of the region is required (see also Ophthalmodiaphanoscopy, page 110).

The *chronic* variety of the disease may occur at any age except before the sixth year, because the sinus is not much developed until after that time of life. It is most frequent between twenty-five and thirty, and commoner in men than in women.

The treatment consists in opening the abscess and washing out the sinus with a bichlorid solution. The incision may be made immediately beneath the superior orbital arch, directly outward, so that the bony wall of the sinus, which is here very thin, may be easily opened, if it has

Fig. 248.—Introduction of drainage-tube after evacuation of abscess caused by ethmoiditis. (A patient in the Philadelphia Polyclinic Hospital.)

Fig. 249.—Ethmoiditis (from a patient in the University Hospital).

not already perforated. A. Knapp prefers that the external incision shall pass along the upper orbital border midway between the eyebrow and the bony orbital margin, and next along the inner wall and side of the nose to the floor of the orbit. This is better, in his opinion, than the Killian incision through the eyebrow. After the wall of the sinus has been perforated the contents of the cavity should be carefully removed. The communication between the sinus and the nose should then be re-established, and a drainage-tube passed from the orbit, through the opening, into the nose, or a gauze drain may be passed from without into the sinus at its nasal angle (Knapp).

2. **Disease of the Ethmoid.**—A common disease of the ethmoid cells is caused by a retention of secretion in them—that is, adopting H. Knapp's phraseology, a *retention-cyst* develops. In these circumstances the growth appears at the upper and inner angle of the orbit,

above and behind the internal canthal ligament, and displaces the eyeball downward and outward. It may not be possible to differentiate this *mucocele* from an exostosis until an exploratory incision is made. With ethmoiditis there may also be tumefaction, especially of the inner third of the lid, imperfect movement of the eyeball with diplopia, severe neuralgic pain, and profuse lacrimation. The last-named symptom may cause the affection to be mistaken for dacryocystitis. In purulent disease of the ethmoid cells the natural escape for the pus is into the nasal cavity, where it can be seen beneath the middle turbinated body, or between this structure and the septa; but, as Bosworth remarks, this is by no means its invariable course. In a large number of cases pus escapes through the os planum into the orbital cavity, giving rise to exophthalmos and *orbital abscess*. The purulent collection should be evacuated by a free incision, so placed as to expose the os planum of the ethmoid. After all necrotic and carious tissue is removed, an opening should be forced into the nose. Through it a drainage-tube should

Fig. 250.—Purulent disease of ethmoid and frontal sinus, with fistulous opening at inner angle of orbit (from a patient in the University Hospital).

be passed, by means of which the cavities can be frequently cleansed with a bichlorid or other antiseptic solution.

Fistula of the orbit, presenting above the internal canthal ligament, may be due to disease of the frontal sinus or of the ethmoid, and particularly to disease of the lacrimal division of the anterior ethmoid cells. Cases of this character are often mistaken for lacrimal disease, and, in fact, they present some of the characteristics of the so-called prelacrimal sac abscess. A cure may be effected by forcing with a strong probe, as Gruening suggested, an opening through the base of the fistula into the nasal cavity, thus facilitating drainage through the nose, or, better, by free exploration of the affected sinuses by means of a Killian or Knapp procedure.

3. **Disease of the Sphenoid Sinus:**—Empyema of the sphenoid sinus may exist alone, or more often in association with suppuration in the ethmoid cells, and may appear in an *acute* or *chronic* form. A diagnosis can often be made by catheterization of the sinus. It is of particular ophthalmologic interest on account of the intimate relation

between the walls of the sphenoid cavity and the optic nerve (the optic nerve may even be free in the sphenoid sinus [Onodi]), and an almost necessary symptom is some form of optic neuritis, either retrobulbar or localized in the nerve-head itself. Optic-nerve disease should always lead the surgeon to take the sphenoid sinus into serious account (see also page 589).

In any case of suspected sinus disease a *stereoscopic x-ray* plate should be prepared; this frequently will give accurate information with respect to the size, condition, and the contents of the accessory nasal sinuses.

Other diseases of this region are *polypi, osteomas,* and *hyperostoses.*

Fig. 251.—Sarcoma of the orbit and postnasal space. (From a patient under the care of Dr. Wm. Zentmayer).

Fig. 252.—Exophthalmos from tumor of antrum which involved the orbit (from a patient in the Jefferson Hospital under the care of Dr. J. Chalmers DaCosta).

4. **Disease of the Antrum.**—Empyema of the antrum is not an uncommon affection, and although it does not belong to the domain of ophthalmology, it is sometimes accompanied by marked ocular signs. In addition to the pain located in the cheek, frequently periodic in character, together with the escape of pus from the antrum, there may be a marked edema of the lids, which, if the disease is of long standing, assumes a positively brawny consistency. Dr. Walter Freeman informs the author that he has seen this edema of the lids when only a few drops of pus have been present in the cavity of the antrum. There may also be chemosis of the conjunctiva and some edema of the optic nerve and overfilling of the retinal veins. A persistent edema of the eyelids not otherwise explained should lead the surgeon to make a thorough examination of the antral cavity. A certain number of cases of lacrimal disease presenting in the ordinary forms of dacryocystitis are connected with antral affections.

Growths in the antrum—*sarcoma*, *fibroma*, and *polypi*—may involve the orbit and produce exophthalmos, or more often displacement of the eyeball upward and outward.

The Ocular Complications of Diseases of the Nasal Accessory Sinuses.—The relation of diseases of the nasal accessory sinuses to diseases of the eye has been referred to in preceding pages in the description of various ocular lesions, notably those which occur in the optic nerve, but for convenience of reference they are redescribed in the following paragraphs:

(1) *Lids and Conjunctiva.*—Edema of the lid is a common symptom of frontal and ethmoidal sinus disease, either the ordinary variety, or else a recurring painful form, fugitive in character and associated with violent headache. Watering of the eye, conjunctival congestion, distinct catarrhal conjunctivitis, and deep-seated scleral congestions, sometimes fugacious, and often accompanied by intense headache, ocular pain and slight edema of the corneal epithelium, have been noted as frequent symptoms of sinusitis, especially in its acute or early stages.

(2) *The Cornea and Uveal Tract.*—Keratitis, corneal ulcers, iritis, uveitis, choroiditis, and vitreous opacities may be due to sinus disease. A form of cyclitis with vitreous opacities, which seems to be due to nasal accessory sinus disease, is described by Kuhnt. (See also page 326.)

(3) *Retina and Optic Nerve.*—The most important group of ocular complications of nasal sinus disease are those in which there is sinusitis without external signs of orbital inflammation, but in which there are optic neuritis, neuroretinitis, retinal thrombosis, and phlebitis, or in which, without marked ophthalmoscopic changes, there is a central scotoma. In some cases a typical acute retrobulbar neuritis arises, with all of the symptoms which have been detailed on page 488, while in others the retrobulbar neuritis manifests its presence by a relative central scotoma, with intact outlines of the visual field; later the scotoma becomes absolute and the field of vision contracts. The scotoma may be unilateral, the more usual condition, or bilateral, and most frequently depends upon disease of the posterior ethmoidal cells or of the sphenoid sinus. Occasionally the scotoma assumes a circular or annular shape. The investigations of Onodi have shown that the optic nerve often is in close relation with these posterior ethmoidal cells, and that the thinness of the intervening wall renders involvement easy, even easier, it is probable, than in the case of the sphenoid, which anatomically may come in close relationship with the nerve and form the inner wall, or the lower and inner wall, of the optic canal. According to Birch-Hirschfeld, the nerve lesions consist in edema, swelling and proliferation of the glia cells, and destruction of the nerve-fibers. These he attributes to venous stasis and also to toxic agencies. In a certain number of cases the ophthalmoscope reveals the usual picture of optic neuritis, or papillitis, with central scotoma, especially, as in a case recorded by A. Knapp, if the anterior ethmoidal cells are infected, and the author has seen elaborate optic neuritis followed by optic nerve atrophy, with extensive disease of the ethmoid, frontal, and sphenoid

sinus, and sphenoid-sinus disease in which the scotoma assumed the form of the so-called hemiopic paracentral scotoma. The visual fields in disease of the sphenoid sinus may exhibit alterations analogous to those in affections of the pituitary body, for example, bitemporal hemianopsia. If the cause of these optic nerve complications is not recognized and speedily removed, either by suitable intranasal drainage, with or without operation, blindness from optic-nerve atrophy is likely to result. An important symptom of posterior accessory sinus disease, described by J. Van der Hoeve, is *enlargement of the blind-spot* for white and colors. This observation has been confirmed by de Kleijn, the author, and a number of others who have investigated the subject. The enlargement of the blind-spot is attributed by Van der Hoeve to involvement of the peripapillary bundle, which may be the first portion affected in retrobulbar neuritis. If no other cause for such increase in the size of the blind-spot can be found, Van der Hoeve considers it to be a symptom which justifies operation on the affected sinus.

(4) *Orbit and Lacrimal Region.*—A mucocele of the ethmoid or frontal sinus may cause mechanical displacement of the orbital contents and exophthalmos, and Birch-Hirschfeld's investigations have shown that nearly 60 per cent. of the cases of orbital inflammation which he has analyzed were due to accessory sinus inflammation. The infection may cause a periostitis over the floor of the frontal sinus or over the os planum, and bring about exophthalmos. This may disappear on treatment, or a *subperiosteal abscess* may develop, which remains encapsulated, or which, by extension, may perforate the skin of the eyelid, leaving an *orbital fistula.* Such periosteal orbital abscesses are frequent in children, the infection being transmitted by the ethmoid labyrinth (A. Knapp). Reber concludes from his studies that infection may reach the orbit (*a*) by direct continuity, (*b*) by way of the venous circulation, and (*c*) by way of the lymphatics. The last-named route has not been positively demonstrated.

Finally, there may be involvement of the orbital structures themselves, resulting in *cellulitis* or *abscess*, either with or without optic nerve inflammation. Extension of antral disease into the orbit is less common and rarely occurs, according to A. Knapp, except through the intermediation of the ethmoidal cells, but both with antral and with ethmoidal infection the symptoms of dacryocystitis may appear, and not infrequently the mistake is made of treating as a dacryocystitis a manifestation of sinusitis.

Other complications which have been recorded are glaucoma and detachment of the retina. The former affection does not occur from sinus disease unless the eye is predisposed to increased intra-ocular tension (Kuhnt). Intense neuralgia, both ciliary and postocular, stubborn asthenopia, and contraction of the visual field have been attributed to the same cause, dependent, according to Kuhnt, on absorptions of toxins from the purulent processes in the sinuses.

Palsy of exterior ocular muscles, notably of the superior oblique, may be caused by sinus disease. The author has observed isolated palsy of

the internus due to ethmoiditis, and certain exterior ocular muscle palsies formerly attributed to rheumatism are doubtless due to sinusitis.

Evidently in the presence of any of these conditions, notably persistent or recurring edema of the lids, fugitive episcleral congestion, retrobulbar neuritis, both acute and chronic, unexplained failure of vision with central scotomas, and stubborn ciliary neuralgia with persistent asthenopia, expert examination of the sinuses is demanded, not only with all of the means at the disposal of rhinologists, but notably with the aid of the *x*-rays, and, in some cases, even if the results of ordinary examination are negative, especially in the presence of the optic nerve complications, exploratory orbital incisions and investigation of the sinuses through them are justified. The diagnostic value of *Van der Hoeve's scotoma* has been explained (see page 590).

Although the ocular manifestations of sinus disease are often marked, it should be remembered, as Sattler points out, that excessive dilatation of the pneumatic sinuses of the skull may pursue an entirely latent course and cause no very decided eye symptoms.

Injuries to the Orbit.—These include fracture of its bony walls, penetrating wounds, the lodgment of foreign bodies, and contusions. The effects of an injury of the orbit depend very much upon the character of the wound and the missile which has produced it. The injury may lead to a phlegmonous inflammation, to hemorrhage within the tissues, and to loss of sight because of rupture of the eyeball or injury of the optic nerve. There are likely to be, according to the circumstances, exophthalmos, displacement of the eyeball, and diplopia. *Hemorrhage into the orbit*, especially beneath Tenon's capsule, will be referred to again as an accident which complicates strabismus operations.

Treatment.—After a penetrating wound a careful search for a foreign body should be made. In a number of instances extraordinary foreign bodies have been found in the orbit, and, curiously enough, very remarkable toleration of the presence of such bodies. If the penetrating wound has cut off the attachment of one of the ocular muscles and the patient is seen soon enough, an endeavor should be made to suture the detached ends. In cases of excessive hemorrhage within the orbit it may be necessary to make an incision and remove the escaped blood. *x*-Ray examination naturally furnishes a means of detecting foreign bodies in the orbit.

Dislocation of the Eyeball.—The eyeball may be dislocated between the lids, which are contracted behind it. It is a rare form of injury. An eyeball may purposely be pried from its socket by means of a thumb thrust into the orbit from the outer side. Luxation of the globe as a self-inflicted injury has been observed among the insane. The result of such an accident may be laceration of the optic nerve and destruction of sight. In other instances the vision has remained unaffected. In certain cases of exophthalmos it is possible to produce this dislocation by pressure upon the globe with the thumbs, the relaxed muscles permitting the eyeball to protrude between the lids. The eye should be replaced and bandaged; it may be necessary to divide the external commissure.

Enophthalmos, or retraction of the eyeball, occurs both as an idiopathic and a traumatic affection. Exophthalmos the result of exhausting diseases is more apparent than real, but a true sinking of the globe, producing an appearance not unlike that caused by a badly fitting artificial eye (Nieden), may follow a traumatism in the neighborhood of the orbit (Fig. 253).

This retraction of the eyeball may immediately follow the injury, or be delayed for days or even months. According to the conditions which are present, it has been ascribed to paralysis of Müller's orbital muscle from lesion of the sympathetic (Schapringer); to atrophy of the retrobulbar cellular tissue caused by trophic nerve disturbance (Beer); to fracture and depression of the orbital bones with cicatricial adhesion or contraction; and to injury of Tenon's capsule and the check ligaments (W. J. Shoemaker). It may be associated with palsy of the inferior oblique (Fuchs, Sachs).

Fig. 253.—Traumatic enophthalmos, patient looking straight forward; sunken appearance, resembling a badly fitting artificial eye, well shown.

Exophthalmos caused by paralysis of the ocular muscles, tenotomies for the relief of strabismus, Graves' disease, orbital disease, orbital growths, and affections of the nasal accessory sinuses has been referred to. It may also occur as the result of irritation of the cervical sympathetic under the influence of certain poisons, notably thyroid extract and paraphenylendiamin, in acromegaly, myelitis, and certain tumors of the brain, notably those which are situated in the neighborhood of the third ventricle and in the middle fossa of the skull. (See also Proptosis, page 88.)

Hemorrhage into the orbit causing exophthalmos takes place in certain diseases—*e. g.*, scorbutus and hemophilia—and has been noted in a *recurring form* in arteriorenal disease. The proptosis in subjects of infantile scurvy is sometimes associated with hemorrhage beneath the skin of the eyelids and the conjunctiva.

Intermittent exophthalmos is a rare affection, about 60 cases being on record. It has been well described in this country by Posey. The characteristic symptoms are a more or less rapid, steadily forward movement of one eye when the head is placed in a dependent position, or when the flow of blood from the head to the trunk is impeded to any extent. In other circumstances the eye usually presents a normal appearance, or, on the subsidence of the exophthalmos, there may be a slight enophthalmos. According to Birch-Hirschfeld, intermittent exophthalmos depends upon a varix of the orbital veins, the origin of which may be congenital, although usually the venous stasis does not take place until later in life, and occurs under the influence of the mechanical factors to which reference has been made.

CHAPTER XXII

OPERATIONS

Ophthalmic surgeons, in so far as the preparation of their hands, gowns, operating-rooms, and surroundings is concerned, naturally follow the strict rules of modern surgery.

Preparation of the Skin of the Region of Operation.—The skin should be treated first with soap and water, then with alcohol, and finally with corrosive sublimate (1 : 2000). These irritating substances must not enter the conjunctival sac, but the face, surface of the closed lids, eyebrows, brow, and scalp should be thus prepared. The ciliary margins should be carefully cleansed with soap and water, followed by bichlorid of mercury (1 : 5000). The parts should be kept covered with a compress of lint soaked in the bichlorid solution until the operation begins. In place of this preparation the eyelids may be cleansed with benzine on a cotton swab, to be followed by a thorough washing with fluid neutral soap. This is the practice in Fuchs' clinic.

The preparation of the conjunctival sac depends upon the nature of the operation (see page 652).

Preparation of the Instruments.—All coarse instruments, such as hooks, scissors, etc., should be boiled for at least ten minutes in the usual manner in a sterilizer.

Sharp instruments—cataract knives, keratomes, cystotomes, etc.—must be cleansed with great caution lest damage be done to their edges. First the edge of the instrument is inspected with a magnifying glass, next the instrument, wrapped in cotton, is put into boiling water for five minutes, and from this transferred to a dish containing absolute alcohol, carefully wiped with the cotton saturated in the alcohol, and finally placed in a tray of sterile water. Just before the operation begins, it is removed from the water, thoroughly dried, laid upon a layer of sterile gauze, and covered with another layer of the same material. As boiling is likely to spoil the edges of sharp instruments, Stroschein believes that it is sufficient to rub them with cotton-wool soaked in a mixture of equal parts of alcohol and ether, and subsequently to wash them in a 5 per cent. solution of carbolic acid.

Dressings.—These must be modified according to circumstances. In plastic operations about the lids the ordinary dressings—that is to say, steam- or heat-sterilized gauze—may be applied, held in place with a sterile gauze roller. Iodoform gauze is occasionally useful in packing the orbit after evisceration, although ordinary sterile gauze yields equally satisfactory results.

If a wet dressing is desired, the fabric may be soaked in bichlorid solution (1 : 5000), saturated boric acid solution, or in a physiologic salt

solution which has been sterilized by boiling, the last preparation being especially valuable if skin-grafting has been employed. Bits of sterile gauze or tightly packed pledgets of cotton wrung out from a 1 : 5000

Fig. 254.—Figure-of-8 of one eye.

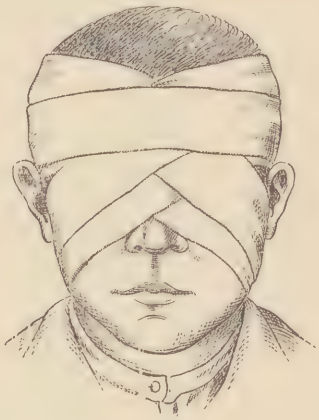


Fig. 255.—Figure-of-8 of both eyes.

bichlorid solution are useful for removing blood, etc., from the area of operation. The various dressings used after cataract extraction, iridectomy, etc., will be described in another section (see page 576).

Fig. 256.—Modified Liebreich's bandage.

Either a single or double gauze bandage may be employed, or a modification of Liebreich's bandage.

Sutures.—These may be of catgut or of silk. In the author's opinion silk is always the preferable material, and black silk is more satisfactory on account of the ease with which it can be detected when the time comes for its removal.

General Anesthesia.—The indications for general anesthesia in ophthalmic surgery are limited. In children or in nervous adults, and for enucleations, eviscerations, etc., blepharoplastic operations, occasionally in advancements of the muscles, and in most cases of glaucoma, general anesthesia is necessary. The surgeon must decide between ether and chloroform.

The author prefers to use the former, as it is safer than chloroform or the mixture of chloroform, ether, and alcohol. Bromid of ethyl has been recommended. The author has not been favorably impressed with this anesthetic. The practice of beginning an anesthesia with nitrous oxid, which is to be continued with ether or chloroform, obviously possesses many advantages. Primary inhalation of ethyl-chlorid is com-

mended by some surgeons. With scopolamin-morphin anesthesia, sometimes employed in surgical operations, the author has had no experience, nor has he had experience with intravenous injections of ether.

Local Anesthesia and Analgesia.—1. **Cocain.**—Hydrochlorate of cocain is usually employed in a 2 or 4 per cent. solution. A 10 per cent. solution has been advised in the operation of curetting lupus and similar growths. General anesthesia is more satisfactory. Cocain causes drying and roughening of the corneal epithelium. This may be partly avoided by keeping the lids closed after each instillation. The drug should not be used too freely, or it may, according to Mellinger, prevent closure of the corneal wound. Gelatin disks impregnated with cocain, as recommended by some surgeons, have no advantage over a solution of the drug. For thorough local anesthesia Haab recommends the application of a thin layer of cocain in crystals. Various fungi grow readily in solutions of this alkaloid, and, indeed, in solutions of any of the alkaloids commonly used in ophthalmic practice. A number of methods of sterilization are employed, namely, sterilization by heat, by the addition of an antiseptic (a 1 : 5000 solution of bichlorid of mercury, 4 per cent. of boric acid, or trikresol, 1 : 1000, as was suggested by Dr. E. A. de Schweinitz, or by the combination of these two methods). The best method, however, is to boil the solution. A number of convenient flasks designed for this purpose are on the market, among the best being those introduced by Dr. Stroschein, of Wurzburg (since improved and modified by Sidler-Huguenin), and the one devised by Llewellyn, of Philadelphia (Fig. 257). The solution is placed in the latter flask and boiled. After the liquid is cool and ready for use, the warmth of the hand causes the fluid to drop from the end of the pipet. If it is desired to preserve the solution after boiling, a portion of one of the antiseptic substances previously mentioned may be added. Boiling is apt to decompose cocain and destroy its anesthetic value.

Fig. 257.—Flask for sterilizing collyria.

2. **Novocain,** although inferior to cocain as an ocular anesthetic, has certain advantages in that its solutions may be sterilized by boiling and it is much less toxic. It may be used in a 1 or 2 per cent. solution, and is employed with advantage in infiltration anesthesia. For this purpose it may be combined with adrenalin.

3. **Eucain** may be obtained in the form of hydrochlorate of eucain "A," which in 2 per cent. solution is an efficient anesthetic, but produces disagreeable congestion of the conjunctiva, and in the form of hydrochlorate of eucain "B," which is related to eucain "A," and also to cocain and tropacocain. It is not decomposed by boiling, and in 2 per cent. solution is an active anesthetic which does not dilate the pupil and is said not to cause clouding of the corneal epithelium.

4. **Holocain.**—A 1 per cent. solution of this drug causes anesthesia in from fifteen seconds to one minute, which lasts for about ten minutes, preceded by a moderate burning sensation. It is an admirable local anesthetic, and its solution does not enlarge the pupil, does not affect accommodation nor increase intra-ocular tension, and is said to possess bactericidal properties (Randolph). It is preferred by many surgeons to cocain in operations on the eyeball, for example, cataract extraction. Its value as an application to corneal ulceration has been described. A mixture of cocain and holocain is also employed.

5. **Acoin.**—This drug is related to caffein and theobromin, and, according to Randolph's experiments, is an active local anesthetic in unirritated eyes in solutions of 1 : 100 and 1 : 300. It has no effect upon accommodation, the size of the pupil, and does not increase intra-ocular tension or cloud the corneal epithelium. In congested eyes even repeated instillations of acoin do not produce satisfactory anesthesia. By some Continental observers it has been advocated as an anesthetic to prevent the pain of subconjunctival injections.

6. **Stovain.**—This drug in 4 per cent. solution, dropped on the conjunctiva, causes smarting, burning, and lacrimation, followed by anesthesia, which lasts for about five minutes. It has little or no influence on the pupil and does not cause paresis of accommodation. In aqueous solution it is not altered by boiling, which renders its sterilization convenient. It has also been used as an injection into the tissues to produce local anesthesia. The author has had no experience with the drug.

7. **Alypin.**—This synthetic compound is a glycerin derivative. A 2 per cent. solution instilled into the conjunctival sac causes slight smarting, some dilatation of the superficial vessels, especially those around the cornea, and anesthesia, which is evident in about one minute (Stephenson). It apparently does not dilate the pupil, and is said to have no influence on accommodation.

8. **Dionin.**—This is a morphin derivative, which produces, a few seconds after its instillation into the conjunctival sac, smarting, burning, stinging, and marked edema of the conjunctiva, especially of that of the bulbar expansion. Occasionally the lid participates in the swelling, and not rarely the "dionin reaction" is severe. Soon the eye establishes immunity, and after a few applications on succeeding days the reaction is little marked and sometimes does not take place. Within twenty minutes the edema of the primary reaction subsides and analgesia appears, which may last for several hours. The drug is a lymphagogue, an analgesic, and probably has some influence in altering and conserving the nutrition of certain tissues, for example, the cornea. Its lymphagogue action is the important therapeutic one. Its indications have been described with the various diseases, and it is especially valuable in the treatment of certain types of ulcerative and parenchymatous keratitis, iridocyclitis, and glaucoma. The author employs the drug in a solution varying in strength from 1 to 5 per cent., according to the indications, from once to four times per diem until immunity is estab-

lished; the drug is now discontinued for three days; at the expiration of this time a modified reaction will again usually appear. If it is urgent that the drug shall be continued after immunity is established, the strength of the drug is increased, but never beyond 10 per cent. It may also be employed in salve or powder. It is an exceedingly valuable remedy; occasionally it produces serious reaction; rarely it aggravates existing conditions, especially if the patients are the subjects of arteriosclerosis and renal disease. The value of the drug may be enhanced by preceding its application with holocain and following it with adrenalin-chlorid. Dionin, although an analgesic, is not a local anesthetic, and in that sense does not belong to those drugs which are used to produce insensitiveness of the tissue with which they come in contact.

With **peronin,** which is related to benzol and morphin, and has an anesthetic as well as a miotic action, and which has been advocated in glaucoma, and with **yohimbin,** which is a local anesthetic and which has been investigated in this country by Claiborne, the author has had no experience.

Infiltration Anesthesia.—In lid operations cocain solution (1 to 2 per cent.) or novocain solution may be injected beneath the skin (holocain cannot be used for this purpose), but probably a more efficacious and safer procedure is the so-called infiltration anesthesia introduced by C. L. Schleich. This consists of an *intracutaneous* (not subcutaneous) injection with a hypodermic syringe, or with one specially devised for the purpose, of a 0.2 per cent. solution of sodium chlorid, which is reinforced by the addition of from $\frac{1}{1000}$ to $\frac{1}{500}$ of 1 per cent. of cocain. The fluid injected produces edema, and the anesthesia is strictly limited to the edematous area.

A mixture of beta-eucain and cocain may be employed for local anesthesia, and very satisfactory results can be produced with beta-eucain and adrenalin-chlorid. Arthur E. J. Barker's solution is as follows:

Pure chlorid of sodium	0.8	gm.
Beta-eucain	0.2	"
Adrenalin-chlorid	0.001	"
Distilled water	100.00	"

The efficiency of this solution can be still further enhanced by adding cocain (H. C. Wood). A 1 per cent. solution of cocain to which 4 minims (0.24 c.c.) of adrenalin (1 : 1000) are added is a useful mixture for local anesthesia (Meller). Pooley recommends the following formula: Alypin, 15½ grains (1 gm.); sodium chlorid, 12 grains (0.78 gm.); adrenalin, 10 minims (0.6 c.c.); distilled water, 3½ ounces (104 c.c.).

Siegrist's Method of Local Anesthesia.—The fluid consists of a 1 or 2 per cent. solution of *novocain*, to which, after sterilization, a few drops of adrenalin (1 : 1000) are added. In enucleation of the eyeball the method is as follows: After the conjunctival sac has been anesthetized in the usual manner, with a curved canula-needle attached to a glass syringe of 2-c.c. capacity, two injections are made

into the posterior part of the orbital cavity behind the eyeball, the needle being inserted on the nasal and temporal sides below the horizontal line, so that it shall not pass through the muscles. Four instead of two deep injections may be made, up, down, in and out, 0.75 c.c. of the fluid being used at each point. Because Seidel has found that this method does not always create satisfactory anesthesia, he has modified it in that with a straight needle he injects 1 to 2 c.c. of the novocain-adrenalin solution around the cornea, 4 mm. from its border. Next four deep injections are made, the needle being inserted upward, downward, outward, and inward over the muscular insertions to a point midway between the optic nerve entrance and the optic foramen. Each injection should contain 1 c.c. of the solution. The immediate results are edema of the lids and exophthalmos, which may be reduced by pressure. Elschnig produces local anesthesia by deep injections in the neighborhood of the ciliary ganglion. This form of anesthesia may be employed in ordinary enucleations, save only that it is not satisfactory in children and nervous persons, nor should it be used if the eyeball is badly shattered or in a state of panophthalmitis.

Local Hemostasis.—For the purpose of producing a hemostatic and astringent action the surgeon may employ various preparations of the *suprarenal capsule*, as originally suggested by Dr. Bates, of New York. The dried and powdered gland (1 part to 10 parts of water) has been used; other preparations are *atrabilin* and *suprarenin*. The most satisfactory preparation (containing the principle isolated by Takamine) is *adrenalin-chlorid*. It is efficacious in a solution of 1 : 10,000, and is active in even weaker solutions. This preparation is used for controlling hemorrhage during slight operations on the eye, for example, tenotomies, excision of pterygia, etc.; for temporarily blanching a congested conjunctiva, and specially, if it is desired to differentiate the types of injection in the different sets of vessels (see page 49); as an adjuvant to the physiologic action of certain remedies, for example, eserin in glaucoma, atropin in iritis, etc.; to enhance the value of certain subcutaneous injections for the purpose of producing local anesthesia and controlling hemorrhage (see page 597); and, finally, as a therapeutic agent, the indications for which have already been given.

OPERATIONS UPON THE EYELIDS

Epilation of the Eyelashes.—Removal of the lashes is performed with forceps known as *cilium forceps* (Fig. 258).

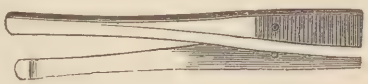


Fig. 258.—Cilium forceps.

The patient being seated in good light, the operator with the fingers of one hand puts the lid upon a stretch, at the same time slightly everting its border. The faulty cilia are firmly seized and pulled out with a quick motion. After those which

are readily seen have been removed, search should be made (with a loupe) for others which may have been broken off, leaving small but irritating ends, and for very fine white hairs which, owing to their lack of color, may escape detection with the unaided eye.

Removal of a Meibomian Cyst.—This may be removed by a conjunctival incision. A sharp scalpel and small curet are required.

The lid is everted, and the discolored patch marking the position of the chalazion is made prominent. This is incised, and the contents are scraped out with the curet.

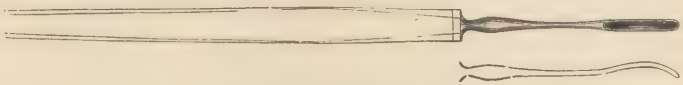


Fig. 259.—Chalazion curet.

The cavity thus formed fills with blood, the absorption of which may be hastened by the use of hot compresses. This operation may leave a slight linear scar in the conjunctiva (Fig. 260).

To avoid this the lid may be grasped between the thumb and forefinger, and by pressure a drop of the jelly-like contents made to appear at the mouth of the Meibomian duct. A few drops of cocain solution are injected by means of a hypodermic syringe the needle of which is pushed into the tumor along the duct. An incision is now made with a Graefe knife, following the course of the needle. A small curet is introduced, and the contents of the cyst are removed (Agnew-Ray). The subsequent blood-clot is absorbed.

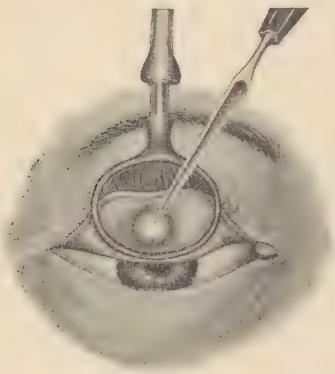


Fig. 260.—Incision of a chalazion (Czermak).

External chalazion should be removed through a skin incision, the lid being steadied in a clamp (Fig. 261), and the cyst dissected from its bed in the ordinary manner.

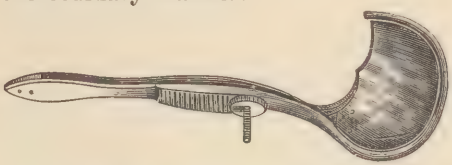


Fig. 261.—Knapp's lid clamp.

Operations for Ptosis.—Before operating for the relief of ptosis the amount of power residing in the levator, or whether it has any activity at all, must be ascertained. The surgeon, standing in front of the patient, firmly depresses the eyebrow with his thumb and requires the subject to open his eyes. Any movement of the lid must be due to the levator, as the pressure on the brow checks the frontalis action; entire failure of lid elevation indicates absence of levator power. If, the frontalis action still being checked and the levator power absent, there is slight elevation of the lid when the eye is rolled upward, it is due to the action of the superior rectus, from which a band passes to the levator tendon.

All operations for the relief of ptosis, and many have been devised, may be gathered into three groups, according to the convenient classification of Grimsdale and Brewerton: (1) Those which shorten the lid or levator; (2) those which utilize the action of the frontalis muscle; (3) those which utilize the action of the superior rectus.

The simplest of the first group, namely, an elliptic excision of a portion of the skin of the lid, is an operation which should not be performed on account of its inefficiency.

Eversbusch's Operation.—The lid is drawn downward and fastened with Knapp's clamp. An incision is now made through the entire width of the lid midway between its margin and the eyebrow, which divides the skin and orbicularis muscle. The edges of the wound are separated for 4 mm. from the underlying tissue above and below, and the tendon, which is thus well exposed, is next included in a loop, with the aid of three double-armed threads passed respectively at the center, the nasal, and the temporal margins. Each needle is now thrust vertically downward between the tarsus and orbicularis, brought out at the free margin of the lid, and securely tied after the wound on the surface of the lid has been closed in the usual manner.

This operation is intended for the relief of imperfect action of the levator and is designed to advance its insertion.

Other operations belonging to this class are advancement of the levator tendon, as designed by Wolff and modified and improved by Elschnig, and excision of a semilunar piece of tarsal cartilage, uniting the edges of the wound with sutures, as advised by Gillet de Grandmont, which is a modification of an operation long ago suggested by Bowman.

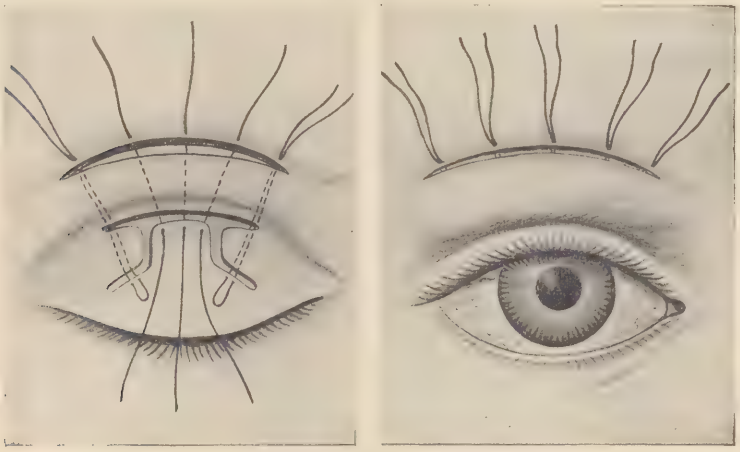
To the second group belong a number of subcutaneous thread or wire operations, which act by establishing a contracting cicatrix or by supplying an artificial tendon.

Pagenstecher's Subcutaneous Thread Operation.—A silk suture armed with two needles is provided. One needle is introduced close to the ciliary border and passed subcutaneously for 2 mm. parallel to the ciliary margin. Next the same needle is re-entered at the point of exit and passed between the tarsus and skin and brought out above the brow. The second needle is introduced at the point of entrance of the first and passed upward beneath the skin to the point of exit of the first above the brow. Finally the sutures are tied.

This method establishes a contracting cicatrix and is suited to cases of incomplete ptosis. The operation was modified and elaborated by the late Mr. Mules, who embedded a fine loop of gold wire in the tarsal cartilage, the two ends from which passed out through the frontalis and which remained and acted as an artificial tendon. Worth uses kangaroo tendon for the same purpose, and Harman "wove-chain" made of fine wire, which is passed subcutaneously from the lid margin to a point below the brow.

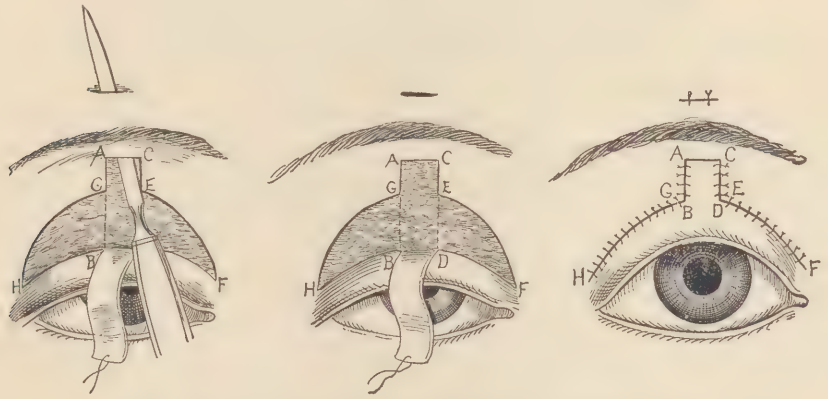
A number of operations have been designed to form a union between the skin of the lid and the frontalis muscle, and among these is the well-known *Panas operation*, which consists essentially in the formation of a small cutaneous flap from the lid, which is passed through an incision

under the brow and is attached to the fibers of the occipitofrontalis muscle, which have been divided by an incision immediately above the brow, and which has cut through all the tissues down to the periosteum. The steps of the operation may be understood by referring to Figs. 262 and 263.



Figs. 262, 263.—Panas' operation for ptosis.

J. O. Tansley has designed a combination of the Panas and Von Graefe operation, or rather, according to M. L. Foster, a modification and improvement of Hunt's operation, with which the author has had gratifying success:



Figs. 264–266.—Tansley-Hunt operation for congenital ptosis.

"Two perpendicular and parallel cuts, *A–B*, *C–D* (Figs. 264–266), ¼ inch apart, are made, and extend from the upper orbital margin to within two lines of the upper edge of the lid. These cuts are united at the upper extremity by a horizontal incision, *A–C*, and the ribbon of tissue is dissected up and permitted to drop down upon a wad of cotton lying on the cheek, which is kept moistened with a warm antiseptic solution. Next, a curved cut is made from *H* to *G* and from *E* to *F*, following the

crease, which shows the upper limit of the tarsal cartilage, and a straight cut is made from *H* to *B* and from *D* to *F*, parallel to and about two lines distant from the lower border of the lid. The skin and the orbicularis embraced within these cuts are now carefully dissected off, leaving the whole tarsal cartilage denuded of tissue. The cut edges *H–G* and *E–F* are united to the cut edges *H–B* and *D–F*, respectively, by interrupted sutures. Next, a narrow Graefe knife is entered at *A–C*, and passed beneath and brought out upon the forehead just above the eyebrow, and slight lateral cuttings are made so as to give room for the passage of the ribbon of skin which has been dissected up at the first stage of the operation. A strong suture placed in the upper edge of this ribbon of skin is used to draw it up into the cut made beneath the eyebrow and bring it out upon the forehead. When it is drawn up sufficiently tight, it is cut off smooth with the forehead and fastened there by two small sutures. Then several sutures are placed from *A* to *G* and *C* to *E*, uniting the edges of the ribbon to the bordering derma." The operation can be readily understood by reference to Figs. 264–266.

In place of the operations just described, the *procedure of Hess* receives the commendation of many surgeons. It consists essentially in making an incision along the shaved brow, through which the lid skin is dissected from the orbicularis muscle, and passing sutures from

Fig. 267.—Ptosis, showing stitches in Tansley-Hunt operation (from a patient in the University Hospital).

without inward through the upper part of the dissected skin of the lid, and carrying them upward underneath the skin to a point above the brow incision, where they are tied. Cicatrization of the wound surface fixes the artificial fold in the upper lid, and the union with the lower part of the frontalis transfers its action to the lid.

Other operations belonging to this group are W. H. Wilder's procedure, who folds upon itself the tarso-orbital fascia and establishes a firm adhesion between the fascia and the frontalis muscle, Fergus' method of attaching a strip of the frontalis to the lid, and Sourdille's modus operandi, by which the levator tendon is fastened to the frontalis.

Finally are those operations which depend for their effect upon a utilization of the action of the superior rectus. Among them the one most frequently employed is that designed by Motais (*Motais' operation*), which consists essentially in attaching a narrow tongue of tissue formed from the center of the tendon of the superior rectus, through an opening in the conjunctival surface of the everted lid, to the upper border of the tarsus, where it is fastened by means of sutures which are brought out

through the tarsus and lid skin and tied on the outer side of the lid. While this operation has certain attractive features, it may be followed by temporary diplopia and depression of the eyeball. If the sutures are tied over the conjunctiva, the knot may cause local irritation. It is warmly commended by H. D. Bruns. If the tendon of the superior rectus is poorly developed, W. T. Shoemaker suggests that the entire tendon of the rectus muscle, in place of a single central strip, shall be fastened to the tarsus in the manner described.

After any of these operations, performed with the usual aseptic precautions, the ordinary dressings should be applied and the sutures removed at the end of a week. The anesthesia may be local or general.

Fig 268.—Result of a Hess operation for ptosis, showing on left side the position of the stitches (from a patient operated upon by Dr. T. B. Holloway).

Tarsorrhaphy.—This operation is designed to shorten an abnormally wide palpebral fissure (*lateral* or *angular tarsorrhaphy*) or to close temporarily the lids over the eyeball (*median tarsorrhaphy*). Lateral tarsorrhaphy is performed as follows:

The external commissure is taken between the thumb and index-finger, the fissure of the lids closed to the required extent, and the line of incision marked with an anilin pencil. A horn spatula or shield is now introduced between the lids, and a flap removed from the free margin of each lid near the external commissure; this must contain all the hair-follicles. The breadth of the flap is 1 mm. and the length about 4 mm. To obtain still firmer union the ciliary margin may be denuded for several millimeters beyond the point of removal of the flap, but in this incision the cilia must not be injured. The edges are approximated by silk sutures. Figure 269 explains the steps: *a* indicates the point of union of the two flap wounds behind the commissure; *b*, *b*, the termination of the flap wounds in the lid-margins; and *c*, *c*, the end of the denudation of the ciliary margins.

Median tarsorrhaphy is accomplished by denuding the ciliary margin of the center of each lid for 4 mm., the lashes being untouched, and approximating the

denuded edge with a mattress suture. The eyeball is thus effectually covered, but the cornea can be inspected if the globe is rotated either inward or outward through the narrowed lid interspace on each side of the central attachment.

Angular tarsorrhaphy is indicated in ectropion in order to raise the angle of the lid, and in lagophthalmos and exophthalmos to improve the unsightly appearance and to protect the cornea.

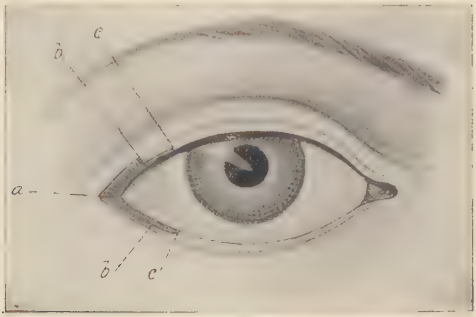


Fig. 269.—Lateral tarsorrhaphy.

Median tarsorrhaphy is suited to those conditions in which the cornea must be protected, for example, in exposure-keratitis, facial palsy, and to prevent ulceration after removal of the Gasserian ganglion.

Canthoplasty (*Blepharotomy*).—This operation is performed to enlarge an abnormally short palpebral fissure.

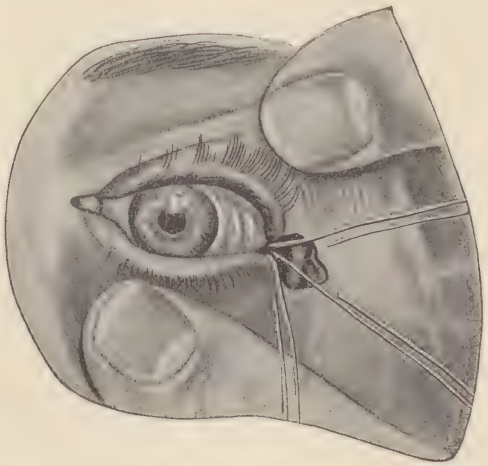


Fig. 270.—Canthoplasty. The stitches ready to be tied (Haab).

One blade of a pair of probe-pointed scissors is introduced behind the external commissure, and the entire thickness of the tissues is divided, making the wound in the skin a little longer than that in the conjunctiva. The wound margins are next separated, and the surgeon loosens the conjunctiva at the apex of the incision and frees it from the underlying tissue. Three sutures are passed, one uniting the ex-

tremity of the conjunctival flap to the center of the skin incision, and one suture above and one below, near the angles of the wound. Division of the external canthus without subsequent introduction of sutures is known as *canthotomy*.

Canthoplasty is frequently performed for the relief of the contracted fissure which follows long-standing granular lids and certain types of chronic blepharitis, and also to lessen the tension on flaps in various types of blepharoplasty. Axenfeld recommends blepharotomy prior to cataract extraction where the palpebral fissure is extremely narrow.

Operations for Trichiasis.—If only a few hairs are involved, the offending lashes should be extracted with cilium forceps in the manner already described.

Fig. 271.—Method of making the intermarginal incision (Czermak).

Electrolysis, as originally suggested by Michel, of St. Louis, may be performed as follows:

A platinum or iridium needle attached to the negative pole of a constant battery is inserted into the follicle of the lash which is to be removed. A sponge electrode attached to the positive pole is applied to the cheek and the current closed. A drop of froth appears around the needle, which should be kept in place for a few seconds and then withdrawn. The lash is easily removed.

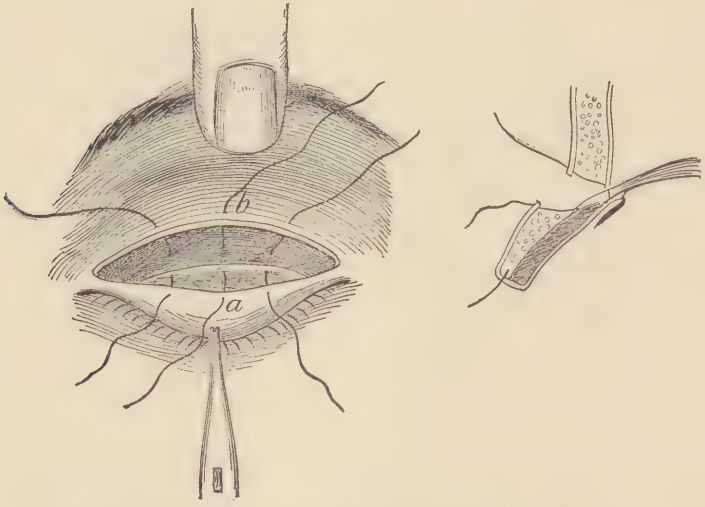
For complete distichiasis some form of transplantation should be employed. The Jaesche-Arlt operation, once often employed, is not satisfactory, and has been abandoned for more rational procedures.

Double-transplantation operations, or, in other words, the manufacture of an artificial lid-border by transplanting a strip of skin to the

intermarginal space, have been practised, especially since Spencer Watson's suggestion.

F. C. Hotz designed the following valuable method:

The lid-border is split by the well-known intermarginal incision (Fig. 271), after which a transverse incision is made through the lid-skin and the orbicularis muscle just below and parallel with the upper line of the tarsal cartilage. The strip of



Figs. 272 and 273.—Operation of Anagnostakis and Hotz.

muscular fibers which covers the upper portion of the cartilage is excised, and the lid-skin is united with the upper border of the cartilage by three sutures. Each suture passes through the edge of the lid-skin, then through the upper border of the cartilage, and finally through the upper edge of the cutaneous wound (Figs. 272, 273). After the sutures are tied, the skin of the lid is drawn upward and fastened to the upper border of the tarsus. By this means a thorough eversion of the anterior edge of the split lid-border is effected, and the intermarginal incision is converted into a gaping wound several millimeters in depth. This groove is filled with a skin-graft, long and

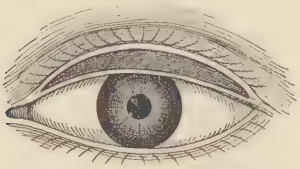


Fig. 274.—Reconstruction of the lid-border (Hotz).

Fig. 275.—Cross-bar entropion forceps.

narrow and somewhat wedge-shaped, which preferably is removed from the integument behind the ear. It should be from $1\frac{1}{2}$ to 2 mm. in width, and of a proper length to fill the opening. The graft is spread out, gently pressed into the groove, and after thorough irrigation with a saline solution both eyes are covered with a compress bandage. During the first two weeks the epidermis of the graft is repeatedly shed, and it is advisable to keep the new lid-border well lubricated with vaselin (Fig. 274).

Because the fine cutaneous hairs in the transplanted flap sometimes irritate the cornea Van Millingen proposed his *tarsocheiloplastic operation*, in which the intermarginal gap is covered with a strip of mucous membrane taken from the inner surface of the under lip.

Operations for Entropion.—Several methods of correcting spasmodic entropion have been referred to on page 178. Gaillard's suture is also useful; the skin of the lid is temporarily shortened by means of a fold caught in one or two sutures. In the *spasmodic entropion* of elderly people the following operation may be performed:

With entropion forceps a strip of skin of suitable width, parallel to the ciliary border of the lid, is pinched up. This strip, together with the subjacent fibers of the orbicularis muscle, is excised. The wound is closed with silk sutures and dressed in the ordinary way. The sutures are removed on the third day.

Instead of excising a horizontal fold of skin, excision of a triangular portion may be performed (Von Graefe). The base of the triangle is placed 3 mm. from the ciliary margin, and the width and length are calculated according to the looseness of the tissues. After the flap is excised the margins are freed and brought together with sutures, but no sutures are applied to the horizontal incision. If necessary, the subjacent tarsal cartilage may be removed.

In organic entropion an operation must be performed which will not merely evert the misplaced border, but also alter the curve of the tarsal cartilage, which usually has become thickened. Two operations will be described:

Burow's Operation.—This operation is designed to relieve entropion of the upper lid following trachoma. It is performed as follows:

The upper lid is thoroughly everted, and the gray-white scar-line (see page 219) which runs parallel with the margin of the lid is exposed At the temporal end of this line an incision is made sufficiently large to admit a fine grooved director, which is now pushed to the nasal side of the lid between the skin and the conjunctiva, care being taken that the point of the director is kept well beneath the cicatricial tissue. The tissue thus elevated is divided in its whole length, either with a sharp scalpel or with narrow scissors. When the operation is completed a blue line equal in length to the line of incision should appear upon the cutaneous surface of the lid. No dressing is required, or, at most, cold compresses to allay the irritation. The cicatricial contraction which ensues everts the incurved border of the lid.

Although the operation is usually primarily successful, its effects generally do not long remain, and recurrence of the entropion takes place. The operation may be repeated several times.

Hotz-Anagnostakis Operation.—This procedure, as it was practised by Dr. Hotz, is described in his own words, as follows:

"A transverse incision from canthus to canthus is made through skin and subjacent tissues, but instead of being made near and parallel with the free border (as in the former methods) the incision in this operation is to follow the *upper* border of the tarsus. It, therefore, describes a slight curve beginning and ending at a point about 2 mm. above the canthus, but being 6 to 8 mm. distant from the free border in the center of the lid. While an assistant is holding the edges of the wound well separated, the surgeon lifts up with forceps and excises with scissors a narrow bundle of the muscular fibers which run transversely along the upper border of the tarsus. Next the sutures, which are to include nothing but the cutaneous wound borders and

the upper border of the tarsus, are inserted. The first suture is placed in the center of the lid; the curved needle, armed with fine black aseptic silk, is passed through the lower wound border; there taken again in the needle-holder, it is boldly thrust through the upper border of the tarsus, and returned through the tarso-orbital fascia just above this border; and finally it is carried through the upper wound border (Fig. 272, *a*, *b*). One similar suture is placed at each side of the central one, and these three stitches are usually sufficient for our purpose—to wit, to draw the skin of the eyelid up toward the upper border of the tarsus and establish a firm union between these parts. This artificial union produces a slight tension of the tarsal skin, which, however, is sufficient to relieve any ordinary degree of entropion.

"But when the lids have been badly contracted—when the palpebral aperture has become unnaturally narrow, or the free border of the lid has become entirely merged into the plane of the conjunctiva—these complicated cases require, in addition to the above operation, such surgical measures as canthotomy, the restoration of the free border either by grooving the tarsus or by grafting" (see description, page 606, Fig. 274).

Fig. 276.—Wilder's double knife.

This is a most satisfactory operation, and the results in the author's experience have been excellent. If grooving the cartilage—that is, cutting a groove or narrow gutter along the center of the tarsus from one end to the other—is combined with the Hotz-Anagnostakis operation, the double knife and clamp devised by W. H. Wilder may be employed (Fig. 276). This knife is also most useful for cutting the narrow graft of skin which may be required to restore the lid-border and which is applied in the manner already described (see page 606).

Although the operations for trichiasis and entropion have been separated in the descriptions, it must be remembered that these two conditions are constantly associated, and hence their surgical treatment in most particulars is identical. Many other operations for the relief of these and other lid affections have been devised, but necessarily the author has described only few standard methods, and especially those which have given him personal satisfaction.

Operations for Ectropion.—If ectropion is associated with relaxation of the tissues, as is often seen in old people, excision of a V-shaped piece of the whole thickness of the lid may be practised. This procedure may be understood by a reference to Figs. 277 and 278. Instead of making the triangular excision, as it is in the illustration, it may be placed at the external canthus, and thus disfiguring scars are avoided.

In place of Adams' operation for shortening the lid-border, Müller's modification of Kuhnt's operation may be practised and can be recommended.

Kuhnt-Müller Operation.—With a broad, triangular knife a deep incision is made into the center of the lid-margin, which divides the lid-substance into two portions, the one containing the conjunctiva and the tarsus, and the other the soft tissues and the skin. From the first portion a triangular piece is removed by the aid of two incisions which should converge toward the fornix (Fig. 279, *A*, *a–c* and *b–c*). The two portions of the lid are next separated toward the external canthus

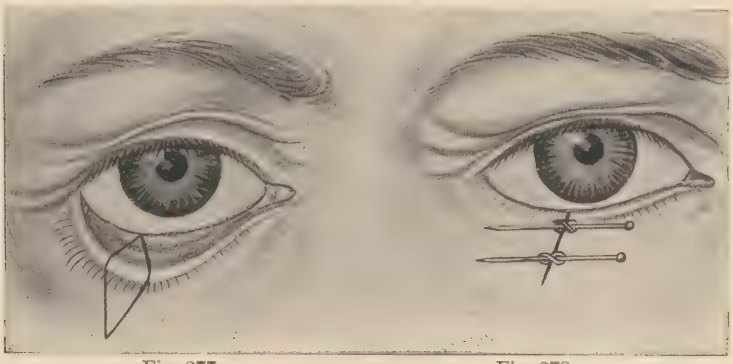


Fig. 277. Fig. 278.

Figs. 277, 278.—Adams' operation for ectropion by excision of a V-shaped piece of the lid.

by carrying the knife from under the margin *b–c* toward *d*. Next, the V-shaped wound of the tarsus is closed with sutures, and the long stretch of the skin margin, *d–a*, is united with the shorter margin, *d–b*, of the tarsus by means of sutures. Their method of application may be understood by examining Fig. 279, *B*. The puckering which occurs after these sutures are tied disappears and the lid margin becomes smooth.

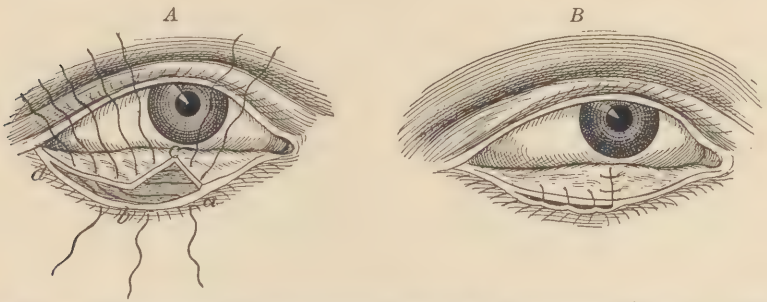


Fig. 279.—*A*, Shortening of lid-border after manner of Kuhnt and Müller; *B*, Kuhnt-Müller operation, final stage (Hotz).

Kuhnt-Szymanowski Operation.—An even more satisfactory operation for the relief of senile ectropion is a combination of the methods of Kuhnt and Szymanowski.

The first step of the operation (Fig. 280) is performed exactly as in the Kuhnt-Müller procedure (see Fig. 279). The next step consists in the excision of a triangular piece of the skin at the external canthus in the manner indicated in the diagram (Fig. 281). The base of this triangle should be somewhat longer than the base of the triangular piece which has been excised from the tarsus. A second incision is carried from the canthus downward and slightly outward, and should be twice as long as

the incision from *a* to *b*. Finally, the two incisions are united in such a manner that the triangular piece of skin can be excised. The skin of the lid is next thoroughly undermined, so that it may be easily drawn outward to cover the defect which has been produced by the excision of the triangular area at the outer canthus. The operation concludes with the insertion of silk sutures in the manner shown in Fig. 282.

Snellen's Suture Operation.—A suture armed with a needle at each end is provided. One needle is entered at the junction of the external and middle third

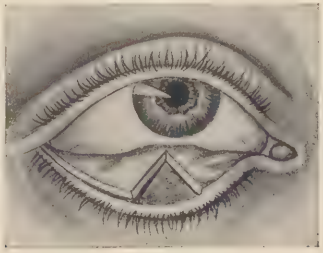


Fig. 280.—Showing the division of the lid into two portions and the excision of the triangle of thickened conjunctiva and tarsus.

close to the posterior border of the tarsus, and is passed down beneath the skin of the lid to a point at the summit of the lower margin of the orbit, and is there brought out. The second needle is entered at a point 5 mm. from the first, and with the other end of the thread is carried down close to the first and parallel with it. The two extremities of the suture are tied upon the cheek over a piece of drainage-tube. The same procedure is repeated with a second double-armed suture, the points of entrance being at the junction of the middle and inner third of the conjunctival

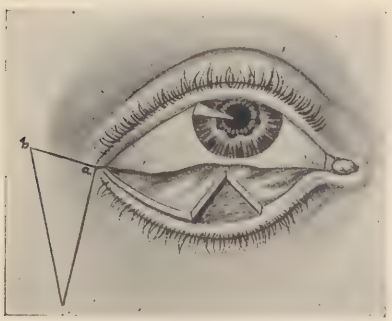


Fig. 281.—Showing the formation of the triangle of skin which is later removed.

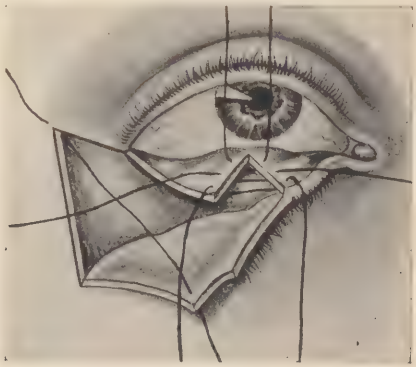


Fig. 282.—Showing the condition after the excision of the triangular piece of skin and the undermining of the lid, which is turned outward. The sutures are in place.

surface. This operation is suited to cases of spastic ectropion. It has been employed in senile ectropion, but has in these circumstances no valuable permanent effect.

Galvanocautery puncture is recommended by S. Lewis Ziegler for the relief of ectropion and entropion. A lid-clamp is adjusted and the galvanocautery point is pushed through the cartilage and quickly

withdrawn. The punctures are made 4 mm. from the lid margin, and separated from each other by an interval of 4 mm. They are made on the conjunctival surface in ectropion, and skin surface in entropion.

Ectropion from the contraction of cicatrices, abscesses, etc., usually requires a plastic operation (*blepharoplasty*) in which the vicious cicatrix is embraced in an incision. If the scar is small, the operation may be done in the manner indicated in Figs. 283, 284—a method which is known as Wharton Jones' operation.

A horn spatula is put into position to protect the eye, and a V-shaped incision is made. The flap is then separated sufficiently to enable the lid to be pushed up into place. The lower part of the wound is drawn together with sutures, thus converting the V into a Y. If possible, the triangular flaps should include the cicatrix which has produced the original trouble.

If the cicatrix is extensive, the operation consists in dissecting out the scar and filling the gap by transplanting a flap of skin from the forehead, temple, or cheek. Many operations of this kind have been

Fig. 283. Fig. 284.

Figs. 283, 284.—Wharton Jones' operation for ectropion.

devised. The site and character of the lesion will in each instance determine the best method of procedure, and it would not be possible in a chapter of the present scope to indicate in detail the numerous ingenious methods which have been devised for the correction of cicatricial ectropion by these blepharoplastic operations, or for the formation of an entirely new lid to replace one that has been destroyed by some disease, such as lupus.

But in order that the student may have an idea of the formation of the flaps for the relief of the deformities just referred to, Figs. 285 to 291, somewhat modified from those in Meyer's excellent treatise, are introduced, and reference to them will explain the essential features of these operations.

During blepharoplastic operations the author is accustomed to irrigate the field of operation frequently with a tepid solution of bichlorid of mercury (1 : 10,000). When the flaps are in place and the sutures, preferably of silk, are adjusted, the whole area is thoroughly

flooded with a sterilized physiologic salt solution. The flaps are now covered with protective, over which several layers of aseptic gauze are placed, care being taken that the gauze itself does not come in con-

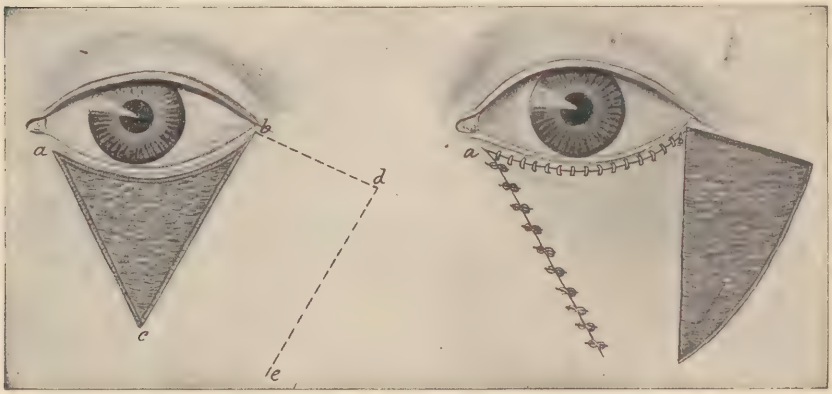


Fig. 285. Fig. 286.

Figs. 285, 286.—Restoration of the lower lid by Dieffenbach's method. The diseased tissue has been removed in a triangular flap, *a–b–c*. This defect is covered by a flap taken from the cheek, indicated by the dotted lines, *b–d–e*, with the result shown in Fig. 286. The remaining gap may be covered with Thiersch grafts.

tact with the flaps, and the whole is secured with several turns of a sterile bandage. This dressing is undisturbed if there are no signs of reaction for seventy-two hours. At the end of this time it is removed,

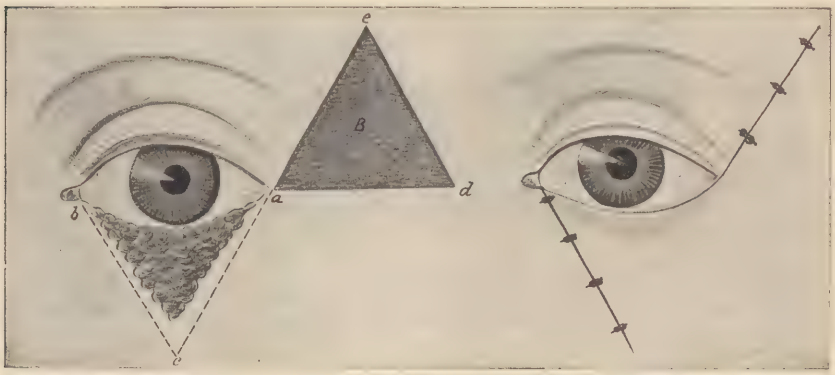


Fig. 287. Fig. 288.

Figs. 287, 288.—Restoration of lower lid by Burow's method. The diseased tissue is removed with the flap *a–b–c*. The horizontal incision is prolonged upon the temple and forms the basis of the triangle *a–d–e*. This flap (*B*) being removed, the cutaneous flap *a–c–d* is dissected up and drawn inward so that the angle *a* is sutured at the point *b*, and *a–d* forms the free border of the lid; *c–a* is now united with *c–b*, and *d–e* with *a–e*, with the result shown in Fig. 288.

any exfoliated epithelium trimmed away, and the edges of the flaps anointed with boric acid ointment. Any gap which remains after the flaps are cut and put in position is subsequently covered with Thiersch

grafts, and the same method is practised if any portion of the flap of skin should slough. If there is breaking down of the epithelium of the flaps, indicating necrosis of the upper layers, the suggestion of Gifford to scrape the area until healthy bleeding tissue is reached, and then apply a large Thiersch graft, may be followed with advantage, although the necessity for this procedure is more likely to occur in cases in which

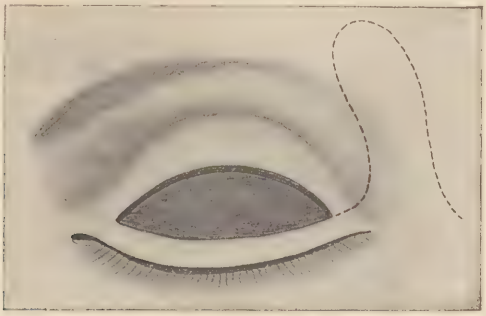


Fig. 289.—Restoration of the upper lid by Fricke's method. The diseased tissue has been removed in an oval flap. The resulting gap is covered by a similarly shaped flap taken from the temple, indicated by the dotted line.

a flap without a pedicle has been transplanted than where the flaps still retain living connection with the surrounding tissues. Sloughing of the flaps should not occur if care is taken to make them large enough to secure them in position without undue tension, and to prepare them in such a manner that they shall receive a sufficient vascular supply through their pedicles.

Fig. 290. Fig. 291.

Figs. 290, 291.—Restoration of the external angle of the lids by Hasner d'Artha's method. The diseased tissue is removed by two elliptic incisions, and the defect covered with a flap taken from the temporal region at *b*, cut in the manner indicated by the dotted line, with the result shown in Fig. 291. The same operation applies to the inner angle, the flap being taken from the nose.

Many disadvantages are associated with blepharoplastic operations, not the least being the extensive scar which may ultimately develop in the position from which the flap was removed. To obviate these a plan originally introduced by Lefort and Wolfe has been practised with success.

Transplantation Without a Pedicle.—This operation consists in transplanting skin without a pedicle from a distant part, preferably the inner side of the arm or the inner side of the thigh. The flap must be about one-third larger than the spot which it is intended to cover, to compensate for subsequent shrinking. It should be shaved down, so as to be as free as possible from subcutaneous connective tissue and fat, and may be held in place by interrupted sutures. The dressing is exactly the same as that previously described.

The primary effect of these operations is sometimes strikingly good, although as time goes on the transplanted tissue is apt to shrink and is said to disappear altogether. The author has been very much gratified with the operation of transplanting skin without a pedicle in selected cases, particularly after removal of small growths from the lower lid, and after burns causing extensive ectropion of the upper lid.

Thiersch's Method of Skin-grafting.—This consists in removing only the upper layer of the skin with a long, wide razor, which is applied flatwise to the inner side of the arm or thigh, and the desired tissue separated by to-and-fro movements with the knife, which is kept flooded with a sterile salt solution. The grafts are transferred directly from the razor to the area which they are to cover. All bleeding must be stopped before they are put in place. They may be dressed in the manner already described or, better, surrounded with a lattice-work of protective strips covered with a compress moistened with salt solution. Thiersch grafts, as already stated, are useful as adjuncts to plastic operations. They may be utilized, however, to supply small defects in the lids and to conceal the deformity of burns around the eyes and face. They have been employed after exenteration of the orbit.

OPERATIONS ON THE CONJUNCTIVA

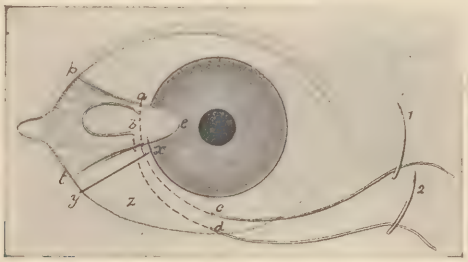
Operations for Pterygium.—(*a*) *Excision.*—The pterygium is seized with a toothed forceps, raised from the surface of the eye, and shaved off with a Beer's knife from its corneal attachment. It is then turned backward, carefully dissected from the underlying tissues, and excised, together with a triangular piece of conjunctiva. This leaves a somewhat diamond-shaped gaping wound in the conjunctiva, which is drawn together with several sutures. If the conjunctiva overlap the corneal margin, two small vertical cuts should be made in it at right angles to the line of excision. After the apex of the pterygium has been separated from the cornea, the vascular subconjunctival tissue must be scraped away down to the sclera; otherwise there will be reattachment. The suggestion of Prince to tear loose the pterygium with a strabismus hook instead of separating the point with a knife is a very good one. Complete excision is not applicable to large nor to fleshy pterygia.

(*b*) *Transplantation* (*Knapp's Method*).—This consists in dividing the corneal attachment, turning the pterygium back, and splitting it from apex to base. The ends are then cut off, and each flap is transplanted into its corresponding upper and lower conjunctival wound, and fixed in position with fine sutures. The exposed surface of the sclerotic is covered by first dissecting up and then drawing together the conjunctiva.

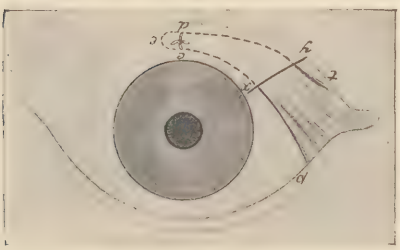
McReynolds' Operation.—This operation, which is a modification of Desmarres' method, gives admirable results, and in the majority of cases, so far as the author's experience is concerned, has proved by far the most satisfactory one in this affection. Dr. McReynolds describes his operation in the following words:

"Grasp completely the neck of the pterygium with strong but narrow fixation forceps. Pass a Graefe knife through the constriction and as close to the globe as possible, and then, with the cutting edge turned toward the cornea, smoothly shave off every particle of the growth from the cornea. With the fixation forceps still hold the pterygium, and with slender straight scissors divide the conjunctiva and subconjunctival tissue along the lower margin of the pterygium, commencing at its neck and extending toward the canthus, a distance of $\frac{1}{4}$ to $\frac{1}{2}$ inch. Still hold the pterygium with the forceps, and separate the body of the growth from the sclera with any small, non-cutting instrument. Now separate well from the sclera the conjunctiva lying below the oblique incision made with scissors. Take a black-silk

thread, armed at each end with small curved needles, and carry both of these needles through the apex of the pterygium from without inward and separated from each other by a sufficient amount of the growth to secure a firm hold. Then carry these cutting needles downward beneath the loosened conjunctiva lying below the oblique incision made by the scissors. The needles, after passing in parallel directions beneath the loosened lower segment of the conjunctiva until they reach the region of the lower fornix, should emerge from beneath the conjunctiva at a distance of about $\frac{1}{8}$ to $\frac{1}{4}$ inch from each other. Next, with the forceps, lift up the loosened lower segment of the conjunctiva and gently exert traction upon the free ends of the threads which have emerged from below, and the pterygium will glide beneath the loosened lower segment of the conjunctiva, and the threads may now be tightened and tied and the surplus portion of the thread cut off, leaving enough to facilitate the removal of the



A



B

Fig. 292.—McReynolds' operation for pterygium: A, Showing needles 1 and 2, which enter the neck of the pterygium *p t e* at *a* and *b*, and then pass beneath the loosened lower segment of conjunctiva *x y z*, and then emerge at *c* and *d* below the cornea; B, showing pterygium *p t e* fixed by a single stitch *c d* beneath the loosened lower segment of conjunctiva *x y z*, while the former side of the growth *p x t* is covered by the normal smooth, stretched, and thinned-out conjunctiva *p x t*.

threads after proper union has occurred. It is extremely important that no incision be made along the upper border of the pterygium; otherwise it would gape and would leave a denuded space when downward traction is made upon the pterygium."

The return of a pterygium after excision is not uncommon; occasionally the second growth is thicker than the primary one, and may exceptionally assume a species of keloid formation. After McReynolds' transplantation operation the author has observed no recurrences, nor has he observed the formation of cysts.

Operations for Symblepharon.—An attempt may be made to remedy this condition by dividing the adhesion and uniting the cut edges of the conjunctiva with sutures, or covering the raw surface left after severing the adhesions with flaps of healthy conjunctiva taken from the unaffected parts of the eyeball (*Teale's operation*), or by dis-

secting back the symblepharon as far as the retrotarsal fold, doubling it upon itself so as to oppose a mucous surface to the globe, and fixing it in this position by means of a ligature which is armed by two needles and passed through the lid from the conjunctiva outward.

The utilization of skin-grafts for the relief of symblepharon is described in the following paragraph:

Transplantation of Rabbit's Conjunctiva and of Thiersch's Grafts.—In cases of extensive adhesion between the ball and the lids the transplantation of rabbit's conjunctiva has been attempted.

In this operation, after the adhesions have been severed, the raw surfaces are covered by a flap of conjunctiva taken from a rabbit's eye, so removed as to be free from all submucous tissue, and somewhat larger than the defect which it is expected to cover. It is better to insert the sutures, with which it is afterward put in place, before its

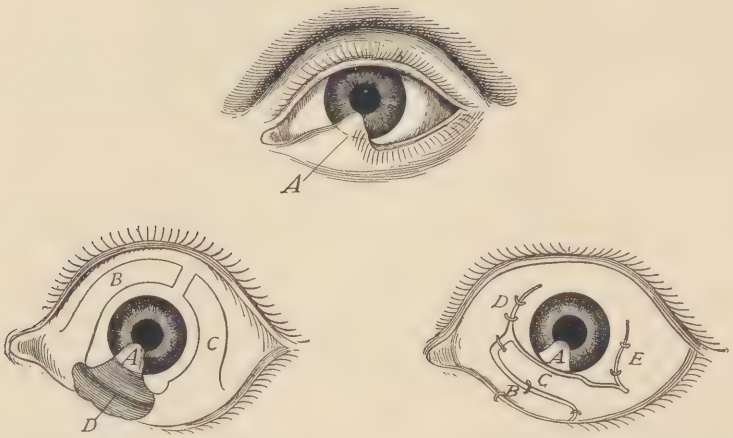


Fig. 293.—Teale's operation for symblepharon (figures from Swanzy). The symblepharon is detached at *A* and removed. Two conjunctival flaps, *B* and *C*, are formed and turned to cover the denuded surface of the eyeball and of the inner side of the lid. The conjunctival gaps are closed by sutures, *D* and *E*.

removal, as they mark the position of the flap, and at the same time give a means by which it may be transferred from the eye of the rabbit to the eye of the patient. It must be kept warm and moist during the process of transferring it. All bleeding must be stopped before the attachment is made. Instead of utilizing the conjunctiva from a rabbit's eye, mucous membrane may be taken from the lip of the patient. In the experience of the author transplantation of mucous membrane is usually an unsatisfactory procedure. A much better method is to utilize Thiersch grafts for this purpose, which are cut in the manner already described on page 614, and which are specially recommended by Hotz, and which the author has used with the greatest satisfaction. The adhesions between the lid and globe are separated in the usual manner, and after all bleeding has stopped the Thiersch graft is put into position. One difficulty is encountered, namely, the

movement of the lid is apt to displace the graft, especially if the denuded area is a large one, and therefore the suggestion of Hotz and May that the transplanted skin should be secured by means of a rigid support —for example, a thin piece of lead or a suitable glass prothesis—is important.

Operations for Trachoma.—On page 224 the operative procedures suited to cases of trachoma are briefly described. Two methods require more extended notice:

Expression (*Knapp's Operation*).—After the patient is etherized the upper lid is everted, seized at the convex border of the tarsus with a pair of fixation forceps, and drawn away from the eye so as to expose thoroughly the whole palpebrobulbar conjunctiva. If the tissue is infiltrated, it may be superficially scarified, preferably with a three-bladed scarifier (Fig. 294). One blade of the roller forceps is pushed deeply between the ocular and palpebral conjunctiva, and the other is applied to the everted surface of the tarsus. The forceps is compressed with some force, drawn forward, and the infiltrated soft substance squeezed out as the cylinders roll over the surfaces of the fold held between it. This maneuver is repeated until all the morbid material has been expressed—in other words, to use Knapp's expression, until the conjunctiva has been thoroughly milked. The lower lid is treated in the same way. During the operation the surfaces should be frequently flooded with a tepid solution of bichlorid of mercury, 1 : 8000, and after the operation cold compresses may be laid on the lid for twenty-four hours.

Fig. 294.—Three-bladed scarifier.

The following day the lids should be everted, and usually a delicate grayish layer of lymph will be found covering the entire area of operation. This should be removed, the swollen mucous membrane exposed, and touched in the ordinary way with a solution of nitrate of silver, 5 to 10 grains (0.324–0.650 gm.) to the ounce (30 c.c.). Each day this treatment should be repeated until the swelling has subsided, when the daily application of a crystal of sulphate of copper is advisable.

The operation should be done thoroughly, care being taken to include the commissural portions of the conjunctiva, and the subsequent local treatment of the case must not be neglected. Expression is especially valuable in cases of spawn-like granulations (follicular trachoma) and diffuse hyaline infiltration. It may be used in cicatricial trachoma when patches of hyaline degeneration are present. If the patient suffers a relapse, as he may, the operation should be repeated. In a somewhat extended experience the author has never seen any save good results from this method of treating granular conjunctivitis. It should never be used in acute trachoma; indeed, usually no form of operative interference is permissible in these circumstances. Some surgeons consider the operation more effective if after the expression a germicide is brushed into the tissues (Weeks).

Grattage.—After the patient is anesthetized the conjunctival surface is exposed in the manner already described. The trachomatous tissue is then deeply scarified, the incisions running parallel to the margin of the lid. The surface is next rubbed with the back of the scalpel, and the conjunctiva vigorously scrubbed with an

ordinary tooth-brush carrying a solution of bichlorid of mercury, 1 : 2000. If the palpebral fissure is very narrow, canthotomy should precede the operation. The after-treatment consists in measures to prevent adhesions between the folds of conjunctiva and the conjunctival culdesac, and the daily application of a sublimate solution of the same strength as that originally used for at least a week following grattage. The subsequent treatment comprises the usual antiseptic lotions and applications until cure is effected.

This operation is applicable to cicatricial trachoma and cases in the second stage of the disease characterized by sclerotic masses of trachomatous tissue. The reaction is sometimes very severe, and the author has not found this method more efficacious than expression preceded by thorough scarification.

Excision of a strip of conjunctiva containing the trachoma granules or of the exposed fornix and afterward closing the wound with sutures is practised by some surgeons and highly commended.

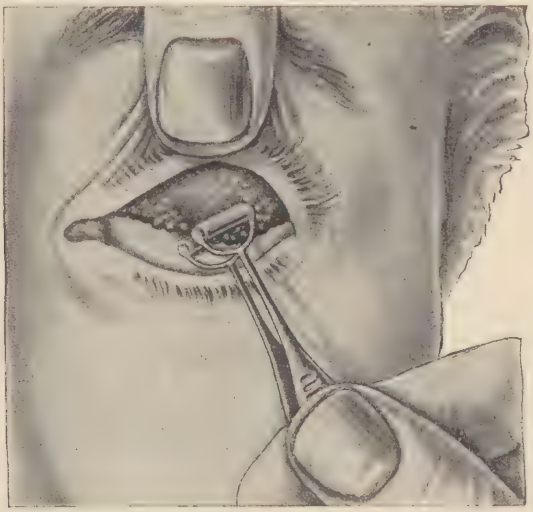


Fig. 295.—Knapp's operation for trachoma (Hansell and Sweet).

Another method includes the removal of a part of the superior tarsal cartilage at the same time that the strip of infiltrated fornix is excised, as was originally recommended by Heisrath. Kuhnt's elaboration of this operation, in that he removes the fold, together with the cartilage and its conjunctiva to within 2.5 mm. of the margin of the lid, is known as the *combined excision*. In cases of chronic trachoma, associated with great infiltration and thickening of the tarsus, Kuhnt recommends *extirpation of the tarsus* with excision of the conjunctiva. With this operation the author has had no experience. The surgical treatment of trachoma by means of the combined excision of Heisrath and the tarsal excision of Kuhnt has been highly recommended in this country by Wootton.[1]

[1] For details of the method of operating, see Archiv. of Ophthalmology, vol. xxxix, 1910, p. 110.

Subconjunctival Injections.—The eye is thoroughly cleansed and anesthetized by the instillation of a 4 per cent. solution of cocain. The patient is required to look strongly downward and inward in order to expose the supero-external portion of the eyeball. Next, the needle of a hypodermic or Pravaz syringe, properly sterilized and charged with the fluid, is introduced very much in the same manner as when an ordinary hypodermic injection is given, well beneath the conjunctiva and away from the cornea. The quantity to be injected depends upon the nature of the case and the character of the fluid employed. If, for example, bichlorid of mercury is used in a strength of 1 : 1000, each division of a Pravaz syringe would contain $\frac{1}{20}$ mg. of the drug. Ordinarily, a solution of bichlorid of mercury, 1 : 2000 or 4000, may be used, and from 4 to 8 minims (0.24 to 0.50 c.c.) injected. Generally, cyanid of mercury is the preferable drug, and may be used in a strength of 1 : 1000 to 5000. The injection may be rendered practically painless by adding a few drops of a 1 per cent. acoin solution to the fluid. Darier's directions are to add one-third of a syringeful of a 1 per cent. solution of acoin to two-thirds of a syringeful of cyanid of mercury, 1 : 1000, and, therefore, obtain a solution of 1 : 1500. Physiologic salt solution is efficient and much less painful. From 15 to 25 minims (0.92–1.54 c.c.) may be injected. If stronger solutions of salt are used, acoin may be added. Solutions of hetol (cynamic acid) in 1 per cent. strength have been advised by Pflüger.

The indications for these injections have been given in connection with the diseases for the relief of which they have been recommended, and, in the experience of the author, they are useful, particularly in various inflammations of the uveal tract, the sclera, and some types of parenchymatous, as well as ulcerated, keratitis. Their value in detachment of the retina, especially solutions of salt, has been described (see page 450).

OPERATIONS ON THE CORNEA

Paracentesis Corneæ.—The local application of cocain is usually sufficient, but in nervous subjects and young children general anesthesia may be necessary. The operation is performed as follows:

The cornea is punctured near its lower margin or, in the case of an ulcer, through its floor with a paracentesis needle constructed with a shoulder to prevent an undue depth of entrance, and inserted at an angle of 45° with the point of contact; or with a broad needle held flatwise, the point being kept well forward so as to avoid wounding

Fig. 296.—Paracentesis needle.

the lens. By rotating the needle slightly on its long axis the lips of the opening are separated and the contents of the aqueous chamber more readily escape. The needle must be withdrawn slowly, lest a sudden gush of aqueous cause prolapse of the iris. The eyeball may be steadied with a spring speculum (see Fig. 298) or fixation forceps (see Fig. 299), provided the former does not put too much pressure on the globe, or the lids may be separated by the surgeon's fingers. If it is necessary to reopen the wound, the probe end of the instrument should be used.

Application of the Actual Cautery.—The indications for this application in corneal disease are given on page 253. If possible, a suit-

able galvanocautery should be employed. If this is not at hand, a platinum probe fixed in a handle similar to the one which is attached to a laryngoscope mirror will suffice. The operation is done as follows:

A few drops of cocain solution are instilled to produce anesthesia, and the probe or the point of the cautery is brought to a red heat, transferred to the area of disease, and all the sloughing material, and particularly the edge of the ulcer, is gently but thoroughly cauterized. It is not necessary to burn beyond the edge of the ulcer into sound tissue. The extent of the ulcerated area, even to the finest spot characterized by loss of epithelium, may be ascertained by the use of fluorescein, but it should be remembered that this drug also colors, but less vividly, diseased epithelium, and hence is apt to stain the epithelium for some distance surrounding the ulcer. Ulcers with much necrotic tissue on them stain yellow. The separation of the lids with a stop speculum is needless; in fact, this is disadvantageous on account of the pressure it exerts upon the eyeball. They may be parted by the hands of the operator himself. After the operation the eye may be washed out with boric acid solution, a drop of atropin instilled, and a bandage applied.

Operation of Saemisch: Saemisch's Section.—The upper lid being raised on an elevator by an assistant, the surgeon proceeds as follows:

The conjunctiva below the cornea is seized with fixation forceps, a cataract knife is entered on one side of the cornea, with its cutting-edge forward, carried across the anterior chamber to the other side of the ulcer, and the section made directly through the diseased area, evacuating the collection of pus in the layers of the cornea and at the bottom of the anterior chamber. If the hypopyon is tenacious, this may be removed by inserting a delicate pair of forceps through the incision and seizing the slough, or it may be washed out with a specially devised syringe. If the pus reaccumulates, the wound should be reopened with a probe and the contents of the anterior chamber again evacuated.

A great objection to this operation is the danger of prolapse of the iris; indeed, owing to the improvement in the treatment of hypopyon-keratitis by means of various local measures and bacterins (see page 255) the operation is much less rarely performed than in former times.

Operations for Staphyloma.—If the measures used to prevent the formation of staphyloma have been unsuccessful (see pages 253 and 262), an operation must be done for its relief. In partial staphyloma vision may sometimes be improved by iridectomy, and even by a double excision of the iris, but very often these measures fail, and then its removal may be necessary.

A useful operation for the reduction of the size of a partial staphyloma is recommended by Berry:

A cataract needle is introduced through the base of the staphyloma and held in one hand. An elliptic piece of the cicatricial tissue of which the staphyloma is composed is then cut out by making one incision at one side of the needle with a cataract knife, and another from the other side, converging toward the first, and in such a manner that the portion held by the needle, and consequently the needle itself, is cut out. The dressing consists of a firmly applied antiseptic bandage, and usually it is necessary to continue the bandage for some time until flattening of the mass has been secured.

After excision of a small staphyloma it is sometimes possible to promote healing by uniting the resulting wound margins with fine silk sutures.

S. Lewis Ziegler's Trefoil or Stellate Keratectomy for the Relief of Anterior Staphyloma.—It may be performed with a punch, thus: A vertical incision is made through the base of the staphyloma, and the inferior blade of a punch is passed into the anterior chamber and beneath the cornea. The punch is closed and the left lateral flap is excised, and in similar manner the right lateral flap is removed. Next the upper margin of the oval wound is grasped at its center by the punch and a vertical piece excised. The trefoil opening is closed with sutures.

De Wecker's Method.—This is suited to complete staphyloma limited to the cornea.

Four sutures should be inserted in the conjunctiva after it has first been carefully detached from the corneal margin almost as far as the equator of the eye. In order to avoid confusion at the moment of tightening the threads, the precaution should be taken of having them of different colors. The removal of the staphyloma is performed by transfixing it through the middle and cutting outward, then seizing the end of the flap thus formed, and removing the rest with scissors. Care must be taken that the lens escapes from the eye. When this is ascertained, the sutures in the conjunctiva are tightened and the conjunctiva drawn over the wound.

In most instances of complete staphyloma, with participation of the sclerotic, the best operation is enucleation or one of its substitutes.

Tattooing the Cornea.—In order to conceal the disfigurement of a dense leukoma it has been suggested to tattoo the white tissue. This is done as follows:

India-ink rubbed up with water into a fine paste is placed close at hand. After the cornea is rendered anesthetic with cocain, the eye is steadied with the fingers, and a drop of the pigment is applied to the surface of the leukoma, and the ink pricked into place with the needles. These needles may be fixed exactly at the same level, precisely as if they were all fastened into a small circular piece of cork, or they may be placed side by side (Fig. 297). Finally, a single needle, somewhat of the type

Fig. 297.—Tattooing needle.

of an ordinary cataract needle, may be employed, and the pigment pricked into the tissue with little stabs made in an oblique direction. According to the late Dr. Noyes, the pigment should be prepared by allowing the India-ink stick to soak for several hours in water until it becomes of the consistence of thick paste. A piece of paste equal to the size of the spot to be colored is then placed upon the leukomatous area and pricked into position with the needles. The tattooing should proceed until a uniform black surface is secured. The excess of pigment can be flooded away with a saturated solution of boric acid. It has been suggested by some surgeons to use variously colored pigments in order to attempt to reproduce the colors of the iris.

The principal operations for *conical cornea*, the method of *removing a foreign body* embedded in the cornea, and the manipulations necessary for closing *scleral wounds* have been described (see pages 279, 280, 293).

OPERATIONS UPON THE IRIS

Iridectomy.—The following instruments are necessary: A stop speculum, fixation forceps, bent keratome, narrow Graefe knife, iris forceps, blunt hook, iris scissors, and horn spatula. The preparation of the patient is described on pages 593, 651, 652. The operation is performed thus:

The patient being in a recumbent position and the eye being under the influence of cocain, unless the case is one of glaucoma, when a general anesthetic is preferable, the surgeon separates the lids by means of a speculum, fixes the eye by seizing with forceps the conjunctiva and subconjunctival tissue at a point directly opposite to that of the proposed section, and introduces the lance-shaped keratome in the following manner: The point of the knife is brought into contact with the apparent corneoscleral margin, or, in some instances, about 1 mm. from the junction of the sclera

Fig. 298.—Eye speculum.

Fig. 299.—Fixing forceps.

Fig. 300.—Keratome.

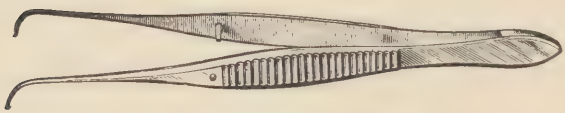


Fig. 301.—Curved iris forceps.

with the cornea, and in a direction at right angles to the cornea, which direction it keeps until the point just penetrates the anterior chamber. The handle is then well depressed, so that the point of the knife shall not wound the iris or lens, while the blade is slowly thrust onward until the section is of the desired extent (see Fig. 305). The knife is then slowly and cautiously withdrawn, with its point well forward toward the posterior surface of the cornea, so as to allow a slow escape of the aqueous humor and to avoid scratching the capsule of the lens.

The *first stage* being completed, the fixation forceps is handed to an assistant, who rotates the globe a little downward, if the section has been made upward, and the surgeon introduces the curved iris forceps, expanding the blades so as to grasp the pupillary margin, cautiously withdrawing the forceps with the included portion of the iris, and snipping off the latter close to the wound by one or two cuts with a delicate pair of curved scissors (Fig. 303). The scissors may be applied in the manner shown in the figure, or they may approach the withdrawn iris from below, the expanded blades being passed over the cornea until they include the iris tissue, which is then excised (see Fig. 306).

If the anterior chamber is very shallow, it is safer to substitute for the lance-shaped instrument a Graefe cataract knife, making a puncture and counterpuncture, and then cutting in the same manner as when the corneal section in cataract is made (see page 654).

If the section of the iris should cause hemorrhage into the anterior chamber, an attempt may be made to remove the blood by separating the lips of the wound with a metal spatula (Fig. 304) and making very cautious pressure on the cornea, but triturating movements carried on to any great extent are done at the risk of bruising the lens and causing cataract. The conjunctival culdesac is disinfected with a warm physiologic salt solution, and the length of the wound, and especially its angles, are inspected to see that the iris is not entangled. Should there be any entanglement of the iris, this must be carefully disengaged with the spatula or olive-pointed probe until the angles of the wound are entirely clear of iris tissue and the pillars of the coloboma perfectly in place. If the wound appears clear, the eye is dressed in the same manner as after cataract extraction (see page 657). One or both eyes may be bandaged, or covered with a pad of aseptic gauze held in place with strips of adhesive plaster. The author prefers to bandage both of them for the first forty-eight hours. Usually the healing is kind, the anterior chamber is quickly restored, and the bandage may be removed at the end of forty-eight hours and the patient directed to wear a shade or dark glass.

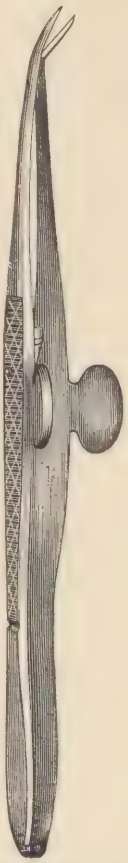


Fig. 303.—Iris scissors.

This, in general terms, describes the method of performing an iridectomy, which, however, may require certain modifications according to the indications and according to the judgment of the operator.

Fig. 302.—Blunt hook.

1. *Position of the Operator.*—The operator may stand behind the patient's head and push the knife from him if he is making an upward section, or he may stand in front of the patient and push the knife toward him in a similarly made section. The latter procedure has been recommended if the anterior chamber is shallow, as the operator can more readily watch the point of the knife. This direction refers to the lance-shaped keratome. The author prefers to stand behind the patient's head.

2. *Point of Entrance of the Keratome.*—This depends upon whether the iridectomy is for optical purposes or for the relief of increased intra-

Fig. 304.—Spatula and probe.

ocular tension. If for the former, its position should be exactly at the apparent corneoscleral border; if for the latter, farther back, about 2 mm. from this position, passing through the sclera. Should a cataract knife be used, preferable always if the anterior chamber is shallow, the position

of the operator and the manipulations are the same as in the extraction of cataract (see page 655).

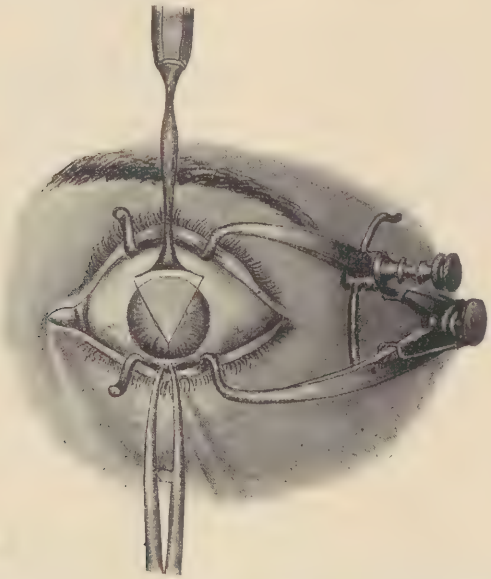


Fig. 305.—Operation of iridectomy; keratome within the anterior chamber.

3. *Position of the Iridectomy.*—If the iridectomy is for optical purposes, the point of selection is governed by the condition of the cornea.

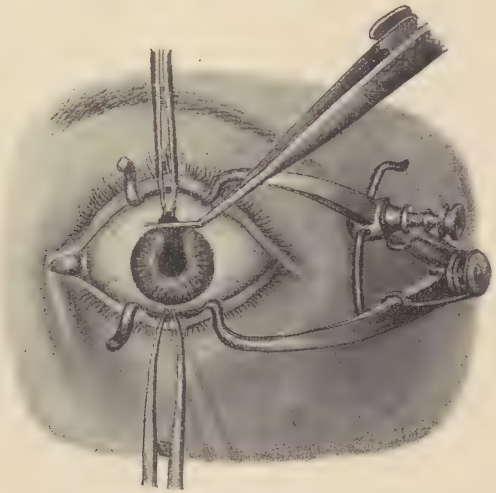


Fig. 306.—Operation of iridectomy; excision of the piece of iris.

The best position for an artificial pupil is inward or inward and downward, other things being equal. In optical iridectomy good results are

obtained and pain lessened by drawing out that portion of iris which is to be excised with a small blunt hook. In place of *optical iridectomy*, as

Fig. 307.—Healed corneal section after iridectomy (from a specimen prepared by Dr. C. M. Hosmer in the author's laboratory).

Fig. 308. Fig. 309. Fig. 310.

Fig. 308.—Broad peripheral iridectomy.
Fig. 309.—Small iridectomy with ciliary border preserved.
Fig. 310.—Narrow iridectomy for optical purposes. (Modified from Swanzy.)

described, Axenfeld performs *precorneal iridotomy*, in which the iris is made to prolapse through a small corneal incision and is incised with a

radial scissor-cut, not excised, and carefully replaced. There is gradual separation of the cut.

If the operation is to restore a pupil to an iris which has been bound down by extensive synechiæ, that portion of the iris is excised which is least attached. Generally it is best to perform the section upward and make a broad iridectomy. The same is true if the operation is performed for a partial cataract, although its exact position must be governed by the condition of the lens.

4. *The Width and Depth of the Coloboma.*—A glance at Figs. 308, 309, and 310 explains three forms of iridectomy: namely, a broad peripheral iridectomy, as in glaucoma; a small iridectomy, with preser-

Fig. 311.—De Wecker's pince-ciseaux.

vation of the ciliary border; and a narrow iridectomy, for instance, for optical purposes.

Iridotomy.—This operation, which is designed to manufacture an artificial pupil, is commonly selected for eyes from which the lens is absent, as after cataract extraction, and in which the pupil has become entirely occluded on account of iritis. It may be performed by simply splitting the fibers of the iris with a knife-needle, the retraction usually affording a sufficient pupil; or a blunt hook (see Fig. 302) may be introduced and the operation converted into a small iridectomy; or a triangular-shaped piece of the iris may be excised with delicate scissors

Fig. 312.—Iritoëctomy (one method): *a–b*, Cornea-iris incision; *a–b*, *b–d*, excision of iris membranes.

Fig. 313.—Iritoëctomy—another procedure (after Czermak).

introduced through a corneal wound (*iritoëctomy*). The method of de Wecker is performed as follows:

A small triangular keratome, preferably fitted with a shoulder, is entered into the apparent corneoscleral margin and pushed on until an incision of about 5 mm. is made. It is then slightly withdrawn and again reinserted, this time causing the point to pierce the iris or the membrane which it is desired to divide. The instrument is now withdrawn, and the delicate forceps scissors of De Wecker is introduced as follows: The instrument is inserted flatwise with closed blades through the wound. One blade is now made to pass through the opening in the iris or membrane and the other in front of it. The blades are now pushed onward as far as necessary, closed after the manner of a pair of scissors, and withdrawn. The cut thus being made across the line in which there is the greatest tension, retraction takes place, and if

the operation is successful, a useful pupil results (*simple iridotomy*). Instead of this procedure, after the narrow keratome which has pierced the cornea and made a small (2 mm.) opening in the iris-membrane is withdrawn, the iris scissors may be introduced, as before described, and two oblique cuts may be made from either extremity of the incision toward the apex of a triangle, forming thus a triangular flap which is removed with forceps (*iritoëctomy*).

A more satisfactory operation than De Wecker's iridotomy or iritoëctomy is one devised by S. Lewis Ziegler, with which procedure the author has had the most gratifying results.

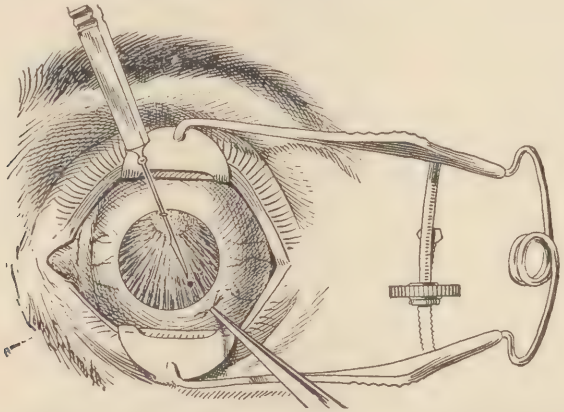


Fig. 314.—Ziegler's V-shaped iridotomy. Knife-needle entered through cornea.

V-shaped Iridotomy (*Ziegler's Operation*).—The instruments required are a speculum, fixation forceps, and Ziegler's modified Hays' knife-needle. It is performed as follows:

First Stage.—With the blade turned on the flat, the knife-needle is entered at the corneoscleral junction, or through the upper part of the cornea (Fig. 314), and passed completely across the anterior chamber to within 3 mm. of the apparent iris periphery. The knife is then turned edge downward, and carried 3 mm. to the left of the vertical plane (Fig. 315).

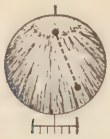


Fig. 315.—Plan of first incision.

Fig. 316.—First incision completed. Plan of second incision.

Fig. 317.—Pupil resulting from V-shaped iridotomy.

Second Stage.—The point is now allowed to rest on the iris-membrane, and with a dart-like thrust the membrane is pierced. Then, without making pressure on the tissue to be cut, the knife is drawn gently up and down with a saw-like motion, until the incision has been carried through the iris tissue from the point of the membrane puncture to just beneath the point of the corneal puncture. This movement is made wholly in a line with the axis of the knife, the shank passing to and fro through the corneal puncture, and the loss of any aqueous being carefully avoided in the manipulation.

Third Stage.—The pressure of the vitreous will now cause the edges of the incision to immediately bulge open into a long oval (Fig. 316) through which the knife-blade is raised upward, until above the iris-membrane, and then swung across the anterior chamber to a corresponding point on the right of the vertical plane, which, owing to the disturbance in the relation of the parts made by the first cut, is now somewhat displaced and the second puncture must be made at least 1 mm. farther over—*i. e.*, 4 mm. to the right of the vertical plane (Fig. 316).

Fourth Stage.—With the knife-point again resting on the membrane, a second puncture is made by the same quick thrust, and the incision rapidly carried forward by the sawing movement to meet the extremity of the first incision, at the apex of the triangle, thus making a *converging* V-shaped cut (Fig. 317). Care must be taken at this point that the pressure of the knife-edge on the tissue shall be most gentle, and that the second incision shall terminate a trifle inside the extremity of the first, in order that the last fiber may be severed and thus allow the apex of the flap to fall down behind the lower part of the iris-membrane. If the flap does not roll back of its own accord it may be pushed downward with the point of the knife. When the operation is completed the knife is again turned on the flat and quickly withdrawn.[1]

Division of Anterior Synechiæ (*W. Lang's Operation*).—This operation is performed with a pair of knives closely resembling Knapp's discission knife-needle. The one is sharp and the other blunt pointed. First, the sharp-pointed instrument is entered through the corneal tissue at a point favorably located for giving a fair lateral movement. It is next withdrawn, and the blunt-pointed knife passed through the same opening across the anterior chamber, with its cutting edge in contact with the synechiæ, which by means of a slight sweeping movement are divided. Occasionally the iris stretches so freely that it is difficult to sever it. Practically no reaction follows the operation, and the subsequent treatment consists in the use of atropin and a compress bandage. If it has been successful, the iris may be dilated and the distorted pupil become round.

This operation, according to Lang, is suited to adhesion of the iris or capsule to the wound after cataract extraction, to traumatic prolapses where a broad width of iris is clamped in the scar, to small adhesions due to perforating wounds or ulcers, and, finally, to large adherent leukomas. In the last group the effects are the least satisfactory.

OPERATIONS UPON THE SCLERA

Sclerotomy (*Anterior Sclerotomy*).—This is an operation first performed by Quaglino, and improved and advocated by de Wecker, which is practised for the relief of glaucoma, and in the hands of some surgeons is made to substitute the operation of iridectomy (see page 391). It is especially recommended in glaucoma simplex with deep anterior chamber, in inflammatory glaucoma with atrophy of the iris, and where iridectomy fails to reduce tension or to relieve the pain of old, blind glaucomatous eyes. It is performed as follows:

A narrow Graefe's cataract knife, or a specially constructed knife known as a *sclerotome*, is passed through the sclerotic, 1 mm. from the margin of the clear cornea in front of the iris, and brought out at a corresponding point on the other side—*i. e.*, the puncture and counterpuncture are placed as if the surgeon intended to form a flap 2 to 2.5 mm. in height out of the upper (or lower) part of the cornea. The puncture and counterpuncture are enlarged with a slight sawing movement of the knife, which is slowly withdrawn before the section is complete, leaving the central quarter of the sclerotic flap, and as much of the conjunctiva as possible, except where punctured, undivided. Thus, at the upper (or lower) margin of the cornea there remains a

[1] The description of this operation is in Dr. Ziegler's own words, and the figures which illustrate it are his, and have been kindly loaned for reproduction.

bridge formed of sclera which connects the parts below it. If prolapse of the iris occurs, replacement should be attempted with a horn spatula. In the event of failure the prolapsed iris must be excised and the sclerotomy converted into an iridectomy. Preceding the operation, eserin should be used to contract the pupil, and this drug must be continued during the process of healing.

Posterior Sclerotomy.—This is performed by entering a Graefe cataract knife at a point between the external and inferior recti muscles, 8 mm. from the corneal margin, and passing the blade through the sclera toward the center of the eyeball to a depth of 4 to 6 mm. As the knife is slowly withdrawn it is made to execute a quarter turn, the effect being the formation of a slight triangular wound, which favors filtration. The operation is employed in hemorrhagic glaucoma, preliminary to iridectomy (see page 388), especially when the anterior chamber is very shallow, and in retinal detachment (see page 450).

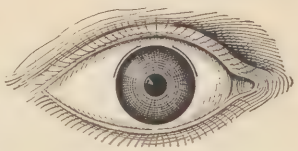


Fig. 318.—Lines of incision in sclerotomy.

Internal sclerotomy was practised by de Wecker and by de Vincentiis under the name of incision of the tissue of the angle of the iris. The incision is similar to anterior sclerotomy, with omission of the counterpuncture, in place of which the arches of the pectinate ligament are incised.

Combined Iridectomy and Sclerectomy (*Lagrange's Operation*).—The operation is performed as follows:

With a Graefe knife the sclera is punctured at a distance of 1 mm. from the limbus and the counterpuncture is made at a corresponding point. The sclera is divided in the iridocorneal angle. In terminating the incision the cutting edge of the blade is directed backward in such a way as to bevel the sclera, and when the knife

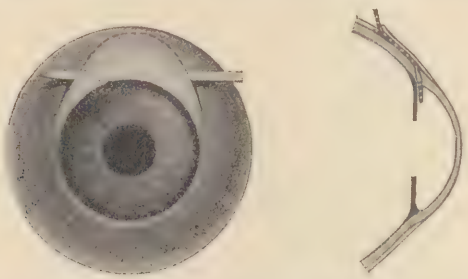


Fig. 319.—Lagrange's operation: Section of the sclera and conjunctiva.

is beneath the conjunctiva a conjunctival flap (about 4 mm. in height) is made. In the second stage of the operation the conjunctival flap is raised, but not cut in any way, and drawn back on the cornea. This maneuver tilts the edge or tongue of the scleral flap upward. This is next removed with a sharp curved pair of scissors, and a sufficiently large piece of the sclera is resected from the exterior lip of the incision. Finally, iridectomy is performed in the usual way, and the flap of conjunctiva detached in the first stage of the operation is replaced, thus covering the defect in the sclera. The steps of the operation are evident by an examination of Figs. 319 to 321. It is not necessary to make a very large flap, 4 to 5 mm. is sufficient.

Lagrange maintains that by means of this sclerecto-iridectomy a communication is made between the chambers of the eye and the perichoroidal space and the subconjunctival cellular tissue. The hyper-

tension of the glaucomatous eye, he believes, is permanently relieved by the establishment of a fistulous track between the anterior chamber and the subconjunctival tissue. Iridectomy, he is willing to admit, cures acute glaucoma, but not the chronic variety. *Simple sclerectomy,*

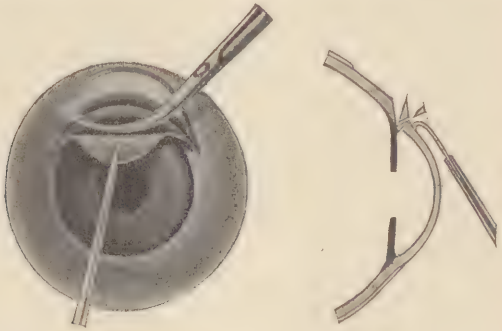


Fig. 320.—Lagrange's operation: Resection of the sclera.

as practised by Lagrange, is performed in precisely the same way; that is, the first two steps of the operation are taken, iridectomy being omitted.

Herbert has devised an operation which, he is satisfied, produces a permeable scar.

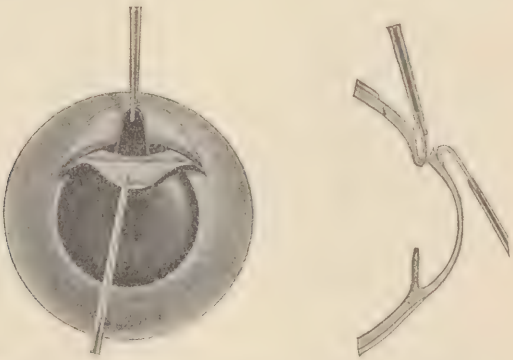


Fig. 321.—Lagrange's operation: Making the iridectomy.

Wedge-isolation Operation (*Herbert's Operation*).—It is thus described by Ballantyne: With a narrow Graefe knife the operator proceeds as if his intention was to form a shallow corneoscleral flap. Puncture and counterpuncture are made close to the margin of the cornea, the knife-point having previously passed through the conjunctiva a little distance above the point of entrance. The upward cut is made with the knife blade bevelled a trifle backward and a bridge of sclera is left undivided. The knife is next brought down and its edge turned forward and a forward cut is made perpendicular to the scleral surface, care being taken not to cut the conjunctiva. This incision forms the lower boundary of the wedge. The knife is now drawn backward and rotated upward so that it lies in the original incision, which is continued upward until the blade emerges through the sclera 1 mm. from

the corneal margin. This completes the isolation of the wedge. A long conjunctival flap is next formed, left attached at its upper extremity, by turning the knife upward and backward. A small, basal iridectomy is made to prevent iris prolapse. This technic has been modified in various ways: thus, the primary incision may be made with a bent keratome and the lateral cuts with a short narrow knife. M. Bishop Harmon has devised "twin scissors" for the purpose of forming the lateral incisions.

Sclerectomy with Punch Forceps (*Holth's Operation*).—With a Graefe knife puncture and counterpuncture are made in the sclerotic 8 mm. from each other and 1 mm. from the limbus. Next, a scleral flap 2.5 mm. high and a conjunctival flap extending to 8 mm. from the limbus are formed. The conjunctiva is now freed from the anterior lip of the scleral flap, raised with forceps, and a piece of the scleral flap, 3 by 1.5 mm., is removed by means of the punch forceps (Holth-Vaclier's, de Lapersonne's, or Ziegler's instrument may be used). A basal or complete iridectomy may be performed, after which the conjunctiva is replaced. The primary incision may be made with a keratome which pierces the conjunctiva 10 mm. above the cornea, enters the sclerotic 2.5 mm. from the limbus, and penetrates obliquely into the anterior chamber.

The important manner in which the results of these operations differ from cystoid cicatrices (see page 390) is that the scar is free from adhesion to the uveal tract. Some difference of opinion exists in regard to their permanent value, and Henderson, although willing to admit that at an early stage such a scar as has been described may be permeable and that filtration may take place, believes it soon becomes impermeable, owing to the ingrowth of epithelium, and, therefore, unless a fistula is made, filtration ceases. Lagrange believes in his operation the fistulous track, before mentioned, is formed. The author has not had experience with Herbert's operation, but has performed the Lagrange operation with satisfaction in cases of chronic (simple) glaucoma. He has not employed it in cases of acute glaucoma, in which form of the disease a well-placed peripheral iridectomy has usually yielded excellent results. According to Meller, the delay in re-formation of the anterior chamber, which is noticeable in some eyes after Lagrange's operation, is due to detachment of the choroid. He regards the operation as unsatisfactory if the intra-ocular tension is very high. He reports 1.3 per cent. of *late infections* in 389 Lagrange operations.

Sclerectomy with a Trephine.—Trephining the sclera for the purpose of relieving the increased intra-ocular tension of glaucoma is not a new procedure. Long ago it was proposed by Argyll Robertson, Strawbridge, Blanco, and Froelich, but their operations differed in many respects from the newer procedures of scleral trephining which have been suggested, particularly by Fergus and by Elliot.

Scleral Trephining (*Fergus' Operation*).—The technic is thus summarized by Ballantyne: A conjunctival flap is dissected up toward the cornea and laid over the corneal surface, while with a Bowman's trephine a small disk of sclera is removed 1 or 2 mm. from the apparent corneal margin. Next, an iris repositor is passed from the trephine opening into the anterior chamber, keeping it in close contact with the sclera and cornea. The conjunctiva is then replaced and stitched into position.

Another operation, and it would seem a more satisfactory one, in which the trephine opening is partly in the sclera and partly in the cornea,

has been designed by Lieut.-Colonel R. H. Elliot. This differs from the Fergus operation, with which Colonel Elliot was unfamiliar when he published his first results, in that a cyclodialysis is not included in its technic, but as now practised a basal or complete iridectomy is part of the procedure and in the position of the trephine opening.

Sclerocorneal Trephining (*Elliot's Operation*).—The description of the technic of this operation, at the author's request, has been written by Lieut.-Colonel Elliot.

1. **The Quadrant of the Eye Selected.**—This should invariably be the upper unless there is some strong contra-indication. The presence of an upward coloboma resulting from a previous iridectomy does not contra-indicate an upward trephining; on the contrary, the operation is made easier thereby.

Fig. 322.—Sclerocorneal trephining: conjunctival flap reflected; trephine opening partly in the cornea and partly in the sclera. (After S. Lewis Ziegler.)

2. **The Flap.**—This should be large. The incision runs roughly concentric with the limbus and ends on either side opposite the highest point of the cornea, and about 8 mm. from its inner and outer sides. The conjunctiva should be seized as high as possible with forceps and drawn well down, while the patient looks strongly toward his feet. One free horizontal cut, followed by a couple of snips at each side, will outline the flap. Only the central area of the flap thus marked is to be dissected up. As we approach the limbus, we should work down to the sclera and should lay the latter bare in the last few millimeters of the wound. We must then clearly define the limbus as a rounded ridge overhanging the adjacent sclera. The area for the application of the trephine must be cleared of all tags of loose tissue. The conjunctival flap should be gently drawn downward by the aid of a blunt instrument, and the cornea is then to be split with the scissor points or with any other fairly sharp instrument. As the dissection proceeds the "dark crescent" of the cornea can be seen as an area convex in outline toward the sclera, and with a straight edge on the corneal side. It is sufficient to split the cornea over an area 1 mm. in depth. This enables a 2-mm. trephine to be applied half on the cornea and half on the sclera.

3. **The Application of the Trephine.**—The trephine (2 mm. in diameter) should be placed *as far forward as possible*, being slid into place from the scleral side, the edge of the flap being meantime keenly watched, so as to avoid buttonholing. A sharp blade is required to make sure of cutting a definite groove in the corneosclera on its first application. Our object should be to make the blade cut through first on its corneal edge, and to ensure this we must slope the upper edge of the instrument a little toward the patient's feet, so that the disk cut out may be hinged on its scleral side. A bead of iris tissue will prolapse through the corneal side of the hole, pushing the disk before it. The disk together with the prolapsed iris is seized in a pair of iris forceps and the two structures are cut with one snip of the scissors, thus performing a sclerecto-iridectomy with a single cut. The amount of disk removed can be graduated according to the needs of the case.

4. **Toilet of Wound.**—The iris is replaced by massage, aided by an irrigator, if necessary, and a dressing is applied. The role of the iridectomy is the same here as it is in cataract extraction; no more, no less. It is safer, in European practice, to secure the flap by means of one or two sutures.

5. **Instillation of Drops.**—From the second or third day onward, provided the tension is down, atropin drops are freely instilled.

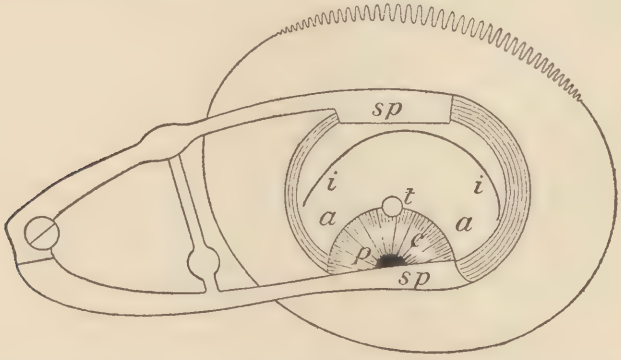


Fig. 323.—*sp*, Speculum; *i*, *i*, incision; *c*, cornea; *t*, trephine hole; *p*, pupil; *a*, *a*, channels in conjunctiva along which filtration fluid passes to enter the main area of the subconjunctival space. (Elliot.)

6. **After-treatment.**—The unoperated eye is opened after twenty-four hours, and both eyes after forty-eight hours. The patient sits up in bed on the second day and moves about the room on the third day.

Naturally, various operators have advised modifications of the procedure; for example, L. Webster Fox (who prefers his type of the Von Hippel trephine) forms the conjunctival flap as in the Van Lint sliding flap in cataract extraction, and McReynolds dissects out a piece of sclerocornea with a knife-point after fixing it with a traction suture. In general terms it has seemed to the author that the most satisfactory results are obtained if Elliot's method is exactly followed. The author prefers a hand-driven trephine; good models are those recommended by Stephenson, Lang, and Elliot.

Indications.—Although many surgeons utilize this operation in acute glaucoma, the author is unconvinced that it is a better procedure than a technically correct iridectomy. It and other methods which secure a so-called "filtering area" are better operations in chronic,

non-congestive glaucoma than iridectomy. If an iridectomy in this disease has failed of its purpose, corneoscleral trephining is preferable to a second iridectomy or sclerotomy. In chronic glaucoma with greatly contracted field it appears to be a safer operation than ordinary iridectomy. It is not a wise procedure if glaucoma is complicated with cataract, but it should be considered in glaucoma secondary to cataract in those eyes in which the vitreous and aqueous chambers are not in communication. It may be performed in absolute glaucoma in the hope of preventing enucleation, but this prevention is not assured. It is not likely to meet with success in glaucoma due to intra-ocular hemorrhage and thrombosis of the central retinal veins; it may be tried in staphyloma and secondary glaucoma, but the outlook is not a brilliant one.

Complications and Causes of Failure.—The chief complications are buttonholing the conjunctival flap, extensive hemorrhage into the anterior chamber, entrance of the scleral button into the aqueous chamber, wounding the lens, loss of vitreous (which should never occur if the trephine opening is correctly placed); intra-ocular hemorrhage, detachment of the choroid, purulent infection, and iritis. After this operation iritis occurs not infrequently, and appears early either as a quiet iritis, with almost no signs of inflammation of the uveal tract, but with the gradual development of soft synechiæ, or arises as a sharp, plastic inflammation at a later period than the first type of the affection. Parker's investigations indicate that iritis is much less apt to develop if complete iridectomy is part of the operative procedure—a significant observation. Its incidence should be prevented by the early use of a mydriatic—the author prefers scopolamin.

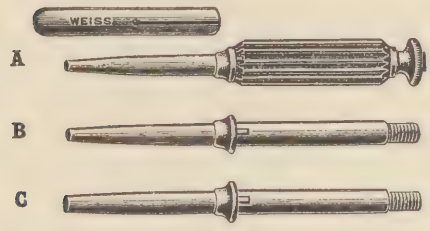


Fig. 324.—Stephenson's sclerectomy trephines.

The causes of failure in this operation, according to Elliot, depend upon forward dislocation of the lens or vitreous body and prolapse of uveal tissue into the trephine hole, which is blocked by proliferated connective tissue either from the episclera or the uvea. In Stephenson's observations the trephine tract was occluded by vascular fibronuclear tissue and iris pigment. A number of *late infections* (from a few weeks to some months after operation) are now on record, the process varying in severity from an infected iridocyclitis and hypopyon to panophthalmitis. Such disasters not peculiar, however, to this operation (see page 414) are due to the entrance of micro-organisms through delicate and invisible fistulas in the conjunctiva covering the trephine opening. After this operation patients with very thinly covered "filtering areas" should be carefully watched and should daily use a collyrium of boric acid and sulphate of zinc (Gifford). Fluorescein should be employed to detect the presence of epithelial defects (Axenfeld, Harms).

Naturally, in a chapter of limited scope numerous operations designed for the relief of glaucoma cannot be described. In Holth's *iridocleisis* the incised iris is incarcerated in the scleral keratome incision and the conjunctival flap allowed to heal over it. To this procedure, entailing iris incision, Borthen objects, and to substitute it has designed an operation called *iridotasis*, the effect being produced by inclusion of a fold of the iris, allowing the posterior surface to coalesce with the subconjunctival tissues, assuring a position of the sphincter external to the section, and with free drainage. In this country this operation is commended by David Harrower. *Zorab's operation* consists essentially of thread drainage of the anterior chamber. A silk thread is introduced into the aqueous chamber and covered with a conjunctival flap. Casey Wood, in this country, has practised this procedure.

Fig. 325.—Corneoscleral trephining; note the filtering area at corneoscleral margin; small peripheral iridectomy.

Cyclodialysis (*Heine's Operation*).—By means of this operation, suggested by Heine in 1905, an endeavor is made to form an artificial communication between the anterior chamber and the suprachoroidal space, but there is no positive proof that after it drainage occurs into this space, although it is possible that a successful cyclodialysis reopens the angle and brings it again into communication with Schlemm's canal. It is performed as follows:

After the reflection of a small conjunctival flap, preferably on the outer side of the eyeball, an opening is made into the sclera with a straight lance, parallel to the corneal margin and from 6 to 8 mm. away from it, without injuring the uveal tissue. This opening should be from 2 to 3 mm. in length, and through it a spatula is introduced with which the ciliary body is separated from the overlying sclera and the instrument gradually pushed through the ligamentum pectinatum into the anterior chamber. Finally, a quadrant of the iris periphery is detached. Occasionally some difficulty is experienced in passing the spatula between the ciliary body and the sclera into the anterior chamber, and in a few instances hemorrhage into this chamber has occurred.

According to Meller, in successful cases reduction of tension is not noticeable until the following day. All increased tension should disappear by the second or, at the latest, by the third day. Occasionally subnormal tension results. If the tension remains low for a week, the ultimate result is likely to be favorable. The operation has proved to be satisfactory in secondary glaucoma due to anterior synechia or sub-

luxation of the lens, in glaucoma following cataract extraction, in advanced cases of chronic glaucoma where iridectomy has failed, and in absolute glaucoma. It is not an operation which is likely to replace iridectomy, and does not seem to the author, who has performed it a number of times with satisfaction in advanced glaucoma, that it should be used for the relief of acute glaucoma or chronic glaucoma of ordinary type.

OPERATIONS ON THE GLOBE AND REMOVAL OF FOREIGN BODIES

Enucleation of the Eyeball.—The following instruments are necessary: A stop speculum, fixation forceps, dissecting forceps, strabismus hook, and a pair of scissors curved on the flat (enucleation scissors).

The lids are held apart with a stop speculum while the surgeon divides the conjunctiva and adjacent fascia with scissors in a circle as close as possible to the margin of the cornea. This is sometimes called "circumcising the cornea." The tendons of the ocular muscles, beginning with the superior rectus, are next successively raised upon a strabismus hook and divided. The eye being made to start forward by inserting the stop speculum somewhat more deeply, the eye is drawn forward, the face of the patient being turned toward the operator, and the curved scissors are introduced on the nasal side between the severed conjunctiva and the freed eyeball, and made to follow the curve of the latter until the optic nerve is reached, where the blades are expanded and the nerve seized and cut squarely off. The attachments of the oblique muscles and the remaining tissue which may cling to the eyeball are then severed. Subsequently the conjunctival wound is closed with a few interrupted sutures.

Hemorrhage is usually not severe, and is readily controlled by pressure. After freely irrigating the socket with a bichlorid solution, it may be dusted with iodoform and a full antiseptic dressing should be applied. In place of general anesthesia, local anesthesia, by means of retrobulbar injections (see page 597), is preferred by some surgeons.

The operation just described is sometimes known as Bonnet's method. The eye may also be removed by what is known as the Vienna method, as follows:

The only instruments necessary are a pair of strong scissors and toothed forceps. The tendon of the internal rectus, together with the overlying conjunctiva, is seized in one grasp with the forceps. It is then divided and the stump retained in the grasp of the instrument. With the scissors the inferior rectus and superior rectus are now divided, together with the overlying conjunctiva. The globe is drawn forward, rotated outward, and the optic nerve divided. The operation is concluded by cutting the external rectus and the two oblique muscles close to the globe. This operation can be rapidly performed. It, however, does not always yield as good a stump as the more slowly performed procedure previously described.

The methods of enucleation just described were almost universally employed until recent years. The technic, however, has been materially improved, chiefly by the various methods of suturing the tendons to the conjunctival bed to prevent their retraction. Suker sutures the severed ends of the recti muscles one to the other, after which the conjunctiva from above and below is brought over the muscle-

stump and fastened with a continuous suture, which also attaches the conjunctival covering to the muscle-stump. H. Schmidt secures each rectus tendon with a catgut suture and makes a slit in the conjunctiva over each muscle, in which then the divided conjunctiva is fastened. The conjunctiva is brought together with a continuous suture. Priestley Smith pinches up a narrow horizontal fold of the conjunctiva over the internal rectus, so as to include the subjacent connective tissue and muscle, and carries a black silk suture through these structures with a curved needle, the suture being tied firmly, but not too tightly. In a similar manner the other straight muscles are attached, after which the enucleation is carried out in the usual manner and the conjunctival aperture closed with one or more vertical sutures.

Freeland Fergus' method of enucleation is as follows:

> The conjunctiva is divided freely over the external rectus so as to expose thoroughly that muscle. As soon as this is done, one blade of the scissors is passed beneath the muscle and it is divided, a small portion of its tendon being left still attached to the sclerotic. This portion is taken hold of with the forceps and the eye is gently rotated toward the inner canthus. When in this position the optic nerve is severed. After the division of the nerve the movement of rotation is continued and all the other tissues are, as they come in view, resected as closely as possible to the sclerotic. A few snips of the scissors suffice. The wound, which is always neat, may be closed with or without the insertion of a gold ball, or with or without suturing together of the muscles, as recommended in the following paragraph.

The author has operated in the following manner with satisfactory results:

> The conjunctiva is divided as close as possible to the corneal margin; each rectus tendon is next seized with forceps, separated from the sclera, and drawn forward to the edge of the cut conjunctiva, where it is fastened with a black silk suture. The eyeball is next enucleated in the ordinary manner, hemorrhage being checked by packing the cavity with a small wad of sterilized gauze. Finally, after removal of the packing, the edges of the conjunctiva are united with interrupted sutures, which are generally placed in a horizontal direction, and which also include the capsule of Tenon. The usual dressing is applied, both eyes being bandaged for twenty-four hours.

The effect of this operation, whereby each rectus tendon is "advanced" to the margin of the conjunctiva and prevented from retracting, is to give a movement to the conjunctival bed very much greater than that which is secured after the ordinary enucleation.

Accidents.—(*a*) *Hemorrhage.*—Occasionally severe hemorrhage occurs during the enucleation of an eyeball, sometimes caused by an anomalous distribution of the vessels. If necessary, the orbit can be packed with antiseptic gauze. The tissues of the orbit may become very much infiltrated with blood and puff out in an alarming manner. The blood-clot, however, will gradually be absorbed, and usually no harm results.

(*b*) *Perforation of the Sclera.*—Sometimes, especially in a ball having very thin walls, the sclera is punctured in the endeavor to cut the optic

nerve. This simply complicates the operation, because it is more difficult to remove a collapsed ball than one which is distended. Should the operator be so unfortunate as to cut through the sclera and leave a portion of it remaining behind, he must proceed to search for the fragment, which can be picked up with forceps, and cut it off, together with the nerve.

(*c*) *Consecutive or Secondary Hemorrhage.*—Occasionally a consecutive or secondary hemorrhage occurs after enucleation. The bandages should be removed, the lids separated, the blood-clot removed, the orbit irrigated with an antiseptic fluid, and, if pressure fails to stop the hemorrhage, a packing of antiseptic gauze should be inserted. Excessive hemorrhage in hemophilic subjects has been checked by the intravenous injection of normal blood-serum.

The *after-treatment* of an enucleation consists in placing the patient in bed, certainly for the first few days. No severe pain ought to follow an enucleation, and decided headache, elevation of temperature, and restlessness may indicate meningeal complication. In a certain number of instances meningitis has followed the operation, especially when it has been performed on an eye within which suppuration is taking place. Under modern methods of operating and with antiseptic precautions this accident is, fortunately, a rare one.

Fig. 326.—Average artificial eye or shell.

Fig. 327.—Solid artificial eye.

Insertion of Artificial Eyes.—An artificial eye may be inserted as early as the second or third week after an enucleation of the eye; indeed, some operators insert it at a much earlier date. For the first week or two the artificial eye should be smaller than that which is a perfect match for the opposite side. The eye may then be exchanged for one which in size is as nearly as possible a match for the fellow-eye. At first the eye may be worn for several hours at a time. Soon it can be worn all day, but it never should be allowed to remain in the socket during the night. It is not necessary to keep an artificial eye in water during the night. It should be washed with a little alcohol and water, and allowed to dry. The wearer of an artificial eye must be cautious that the enamel is always smooth.

In order to insert an artificial eye, the upper eyelid is seized between the fingers of the left hand and drawn gently down and out, and the larger end of the shell is inserted vertically beneath it, then brought to a horizontal direction, while at the same time the lower lid is pulled down, when the shell slips into place. In order to remove an artificial eye, the head of a large pin is inserted beneath its lower margin, the lower lid being at the same time depressed, while the eye is tipped upward and forward, when the pressure of the upper lid will force it out. Very soon

patients become exceedingly expert in taking out and introducing artificial eyes, and do not require the aid of a pin in making the manipulation just described.

One of the chief objections to the shell-shaped prosthesis, or artificial eye, is the fact that in its hollow undersurface tears and mucus may accumulate, while its thin edges may bruise the conjunctival bed. To obviate this difficulty the so-called "reformed artificial eye" has been introduced, largely through the efforts of Professor Snellen, which consists of a double-walled shell, or sometimes of a solid eye, the smooth rounded contour of which neutralizes the objections to the thin edges of the old-fashioned shells. The movements of an eye of this character, placed in the socket after a properly performed enucleation with suture of the tendons or implantation of a gold sphere in Tenon's capsule, are nearly as extensive as those which follow Mules' operation.

Instead of the operation of enucleation, certain substitutes have been proposed, the most important of which are:

Evisceration of the Eyeball.—This consists in an evacuation of the contents of the eye, the sclerotic being unmolested, and closure of the scleroconjunctival wound with sutures, thus forming a movable stump for the artificial eye.

The instruments required for the operation are a speculum, fixation forceps, a narrow knife, a pair of scissors, and an evisceration spoon. It is performed as follows:

The speculum being introduced, the conjunctiva is loosened around the cornea; the anterior chamber is transfixed with the knife on a level with the horizontal meridian, the lower portion of the cornea separated, the flap seized with forceps, and the remainder of the cornea cut away at the corneoscleral margin. With the evisceration scoop the contents of the globe are thoroughly and cleanly evacuated. The cavity of the globe is wiped out with sterilized cotton-wool, and all bleeding is stopped. The edges of the conjunctiva are united by means of a suture similar to the string which draws shut a tobacco-pouch—a suture sometimes called the *tobacco-pouch suture*, or by interrupted sutures. These may include the conjunctiva alone, unless this is very much macerated, when it may be necessary to include the sclera. The author is accustomed to suture both the sclera and the conjunctiva. The evisceration may be accomplished without sacrificing the cornea provided this membrane is not infected (Gifford).

Considerable pain may follow the operation, together with edema and swelling of the surrounding tissues. In order to avoid this, it has been recommended to introduce a horse-hair drain, and Prince has suggested wiping out the cavity with carbolic acid in order to allay the pain.

The chief indication for evisceration is panophthalmitis (see also page 359), although it may also meet the indications which are mentioned below in connection with Mules' operation. Evisceration is contraindicated by sympathetic inflammation or irritation, malignant disease, and much shrunken eyeballs. Although the stump after evisceration is primarily more voluminous than that which is secured after an enucleation, subsequent shrinking of this stump ultimately renders

the cosmetic effect of the operation no better than that which is secured by a properly performed enucleation, while its inconveniences are much greater.

Evisceration of the Eyeball, with Insertion of an Artificial Vitreous.—Mules' Operation.—Mules modified the operation of evisceration by the introduction of a glass ball into the cavity of the sclera. The operation is performed as follows:

After general anesthesia a stop speculum is introduced, and the conjunctiva dissected from the corneoscleral attachment in all directions to the equator of the ball without disturbing the muscles. The cornea and 1 mm. of the scleral margin are removed in the manner described under evisceration. Next the contents of the globe are emptied by any convenient method, a small evisceration scoop being a satisfactory instrument. Great care must be taken to remove the entire contents, leaving a perfectly clean, white sclera. Hemorrhage is controlled by packing the scleral cavity with sterilized gauze, and by frequently irrigating it with a tepid solution of bichlorid of mercury (1 : 5000). A glass or gold sphere (gold is preferable), of such size that it may be introduced within the scleral cup without difficulty, is selected, its introduction being facilitated by slitting the sclera vertically for about 4 mm. at the upper and lower margins of the opening. The introduction of the sphere is further facilitated by the use of an instrument specially devised by Mules for this purpose. The concluding steps of the operation consist in stitching the sclera vertically, the conjunctiva horizontally, and applying a full antiseptic dressing. The patient should be confined to bed for at least four or five days. Considerable reaction may follow, and marked chemosis of the conjunctiva. This may be controlled by the continuous application of cold, and probably be avoided by not removing the bandage for forty-eight or even seventy-two hours. Mules recommended that the sutures should be of catgut; the author prefers silk sutures.

The chief *indications* for this operation are ruptured or injured eyeballs, provided the sclera is not too much lacerated, and the accident of recent date; staphyloma of the cornea and sclera, or complete leukoma; absolute glaucoma; buphthalmos; and non-traumatic iridocyclitis. The chief *contraindications* are suppuration of the eyeball; morbid growths; much shrunken eyeballs, the contents of which have undergone bony or calcareous change; sympathetic ophthalmitis, sympathetic irritation, and pathologic conditions of the eyeball which are likely to produce either of the last-named affections; extensive injuries of the eyeball, with much bruising and laceration of the sclera; dacryocystitis; and ocular conditions demanding enucleation or its equivalent in very old persons.

Implantation of an Artificial Globe in Tenon's Capsule After Removal of the Eyeball (*Frost-Lang Operation*).—The eyeball is enucleated in the ordinary manner, and, after all bleeding has been checked, a gold or glass sphere is inserted within Tenon's capsule. The capsule and conjunctiva are next sutured over the artificial globe with silk sutures, the tendons of the ocular muscles having previously been secured by one of the methods described under Enucleation. The subsequent treatment is the same as that suited to Mules' operation.

Sponge-grafting into the orbit for the support of an artificial eye has been suggested by Claiborne and Belt, and Suker wraps the glass sphere in a layer of sponge before its implantation.

A number of surgeons have recommended, in place of the insertion of a glass or gold sphere within Tenon's capsule, the use of paraffin spheres, and admirable results have been reported, especially by Charles Nelson Spratt. He uses a suitably sized paraffin sphere and places it within Tenon's capsule. Following this, the superior rectus is sutured to the inferior by a mattress suture, and the two lateral recti by a similar suture. Tenon's capsule is closed over the globe by means of a catgut purse-string suture. Finally, the conjunctiva is closed with a similar suture.

Fat implantation into Tenon's capsule after enucleation of the eyeball, originally proposed and practised by Barraquez, came into prominent notice after Bartels called attention to the operation in 1908. The eyeball is enucleated, the recti muscles being secured in the manner already described. A mass of fat taken from the abdominal wall is inserted into Tenon's capsule so as to fill, but not to overstretch, it. The muscles are sutured crosswise or by means of a purse-string suture, and next the cut conjunctiva is united with stitches. Either catgut or silk may be employed. The cosmetic result is said to be good.

L. Webster Fox has devised an operation which consists of *implantation of a glass or gold ball into the orbit after remote enucleation of an eyeball.* It is performed as follows:

> If the operation is to be performed on the right orbit, the eyelids are separated by a speculum, the conjunctiva is grasped up and in above the inner canthus, and the tissues are well pulled out. Next, a Beer's knife or curved keratome is passed through the tissues somewhat obliquely and well down into the orbit, and an opening made large enough for the insertion of the globe behind the tissues. This opening may be enlarged with curved scissors to the desired size. When ready, a gold ball is inserted through the opening, which is closed with two stitches and over which a shell is placed, modeled after an artificial eye. The eyelids are then closed over this shell, which is left in place for twenty-four hours. The stitches are taken out on the third day. If the operation is to be performed on the left orbit, the incision is made up and out above the external rectus muscle and the dissection carried out as above described.

Opticociliary neurotomy and neurectomy have been employed as substitutes for enucleation, and are still performed by some surgeons, but in the opinion of the author they are rarely to be recommended.

Operations for Prosthesis in Cases of Cicatricial Orbit.—Contraction of the conjunctival sac and the formation of cicatricial bands, especially after lime-burns and trachoma, may render the introduction of an artificial eye impossible. The same state of affairs may be brought about by the neglect to wear a suitable eye. Numerous operations have been devised to enlarge the socket under these circumstances, mere incisions of the contracting bands being entirely insufficient. If there is complete closure, or practically complete closure, of the orbital cavity, the most satisfactory results are reached by dissecting loose the attachments of the lid to the contracted orbital tissues and preventing readhesion by the introduction of grafts of skin—either a Wolfe-Lefort flap or a Thiersch graft. It is usually best to restore only

one culdesac at a time, and after the flaps are in place their position in the culdesac should be maintained by the insertion of a plate, which, as John Weeks suggests (and he has elaborated one of the best of these operations), may be made of flexible rubber, such as is used by dentists. If the cavity of the orbit is not entirely obliterated, it is sometimes possible, after dividing the cicatricial bands, to form a socket, in which later an artificial eye may be placed, by introducing lead plates, which are bent to conform to the size of the cavity, and from time to time increased in size. The plate gradually creates a groove in the dense tissue, into which afterward the margin of the artificial eye is fitted.

Removal of Metallic Foreign Bodies from the Interior of the Eye.—For this purpose, as has already been noted on page 295, a giant magnet may be employed (the Haab pattern), and the body drawn into

Fig. 328.—Showing the use of the large magnet in extracting an iron spicule from the eye (Haab).

the anterior chamber, or, having been properly localized by means of the *x*-rays, it is removed by means of a large magnet or one of more moderate size (the Sweet model), through a suitably placed scleral incision which directly overlies the position of the metal. The extension-point of the magnet, however, does not enter the sclera.

Haab's Operation.—This may be performed as follows, according to this distinguished operator's directions:

After the usual aseptic preparations and thorough cocain anesthesia of the eye, the operator assumes one of the two positions shown in the accompanying figures.

In the majority of cases—that is, in all those in which a small- to a medium-sized splinter is probably present—the center of the cornea should be placed exactly opposite the pole of the magnet. If the presence of a large splinter is suspected the pole of the magnet should first be allowed to act at some distance from the eye. The patient is told to look in the direction of the pole of the magnet. The first closure of

the current may bring the foreign body behind the iris. If it does not, the current must be repeatedly opened and closed. If now there is no bulging of the iris, more lateral portions of the cornea are successively brought opposite the pole, but the region of the ciliary body must be scrupulously avoided. To draw the splinter forward, from behind the iris, through the pupil into the anterior chamber is not always an easy matter, although, if it is smooth, it usually comes without difficulty. Occasionally iridectomy is necessary, although Haab has not found this requisite in his personal experience. According to Lang's suggestion, a smooth steel spatula, attached to the magnet, may be carried through a corneal incision behind the iris where the splinter is lodged. In uncomplicated cases after the splinter reaches the anterior chamber it may be removed through a suitable corneal incision by introducing the extension point of a small magnet, although Haab himself finishes the operation with the large magnet. Each case must be carefully considered and the technic varied according to the conditions.

The author's views in regard to the method of removing foreign bodies have already been given on page 295.

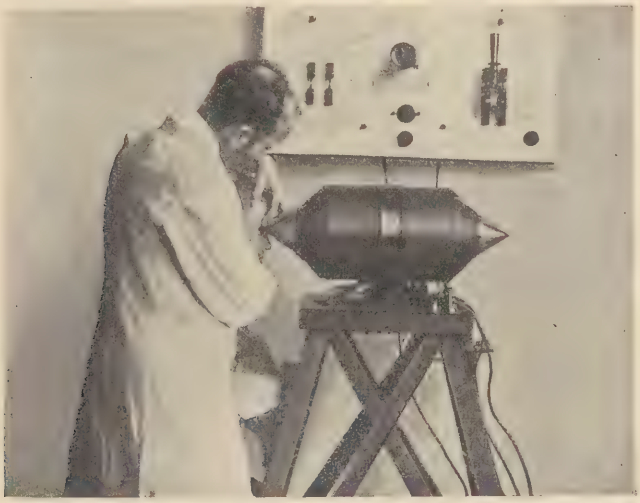


Fig. 329.—Showing the use of the large magnet in extracting an iron spicule from the eye (Haab).

Extirpation of the Whole Contents of the Orbit (*Exenteration*).—This is the operation necessary in certain cases of malignant disease.

The eyeball having been removed in the ordinary way, an incision is made through the outer commissure to the edge of the orbit. The lids having been widely separated, the tissues back of them and the periosteum within the orbital margin are divided with a scalpel. Next, the periosteum is separated to the apex of the orbital cavity, where the entire mass of tissue is detached with strong curved scissors or other suitable instrument. Bleeding, which is sometimes considerable, may be checked by pressure or, if necessary, by the actual cautery. The cavity is loosely packed with iodoform or ordinary sterile gauze, one strand of which may be left at the outer commissure for drainage. Next, the ciliary borders, including all hair-follicles, is removed, and the lids, thus prepared, are sutured together up to the point where the gauze strand protrudes. The packing should be removed at the end of twenty-four hours. In favorable cases the lid-skin will be retracted inward and completely line the socket. Thiersch grafts are also used for this purpose. It may

Fig. 330.—Spindle-cell sarcoma of the orbit which was removed, with preservation of the functions of the eye, through an incision beneath the entire orbital arch.

Fig. 331.—Result after removal of sarcoma of orbit.

happen that the eyeball is so involved with the malignant disease which is present that its extirpation as the first step of the operation is not feasible. The operator then proceeds as before described, removing the eyeball with the entire mass of tissue.

Removal of Tumors and Cysts from the Orbit.—Tumors in the anterior portions of the orbit may be reached by an incision similar to that already described in connection with deep-seated purulent pockets (see page 577), and the growth removed by an ordinary dissection. Occasionally, in favorable situations, such growths may be reached by a dissection through the conjunctiva. If the growth is an angioma, and is encapsulated, it may often be removed in similar manner by a slow dissection, without much loss of blood. If non-encapsulated, and especially if it protrudes and involves the skin of the lid and brow, it is a much more difficult procedure. To a certain extent the hemorrhage can be controlled, as Knapp suggests, by pushing a horn spatula beneath the upper lid, between the eyeball and the orbit, which may be manipulated to act as a controller of hemorrhage, while the dissection proceeds from the skin surface. Although the main body of the angioma may thus be removed, it is often impossible, without sacrifice of too much tissue, to extirpate those portions of it which involve the skin of the eyelids and eyebrow. These, however, may disappear later, or may be treated by electrolysis. Recent investigations indicate that they may be successfully treated by applications of liquid air. Sometimes encapsulated sarcomas, endotheliomas, and certain non-malignant growths, especially in the anterior portion of the orbit, may be reached without sacrificing the eyeball, according to a method advocated by Lagrange and Knapp, namely, first severing, if, for example, the growth is on the inner side, the internal and perhaps the inferior rectus, which are secured with threads, next separating the conjunctiva, and gradually dissecting out the growth through the opening thus made. After controlling the hemorrhage the severed recti muscles are sewed in place exactly as in the operation of advancement. Again, the growth may be reached by means of a dissection which begins with an incision which extends beneath the entire length of the orbital arch and is continued slightly downward and outward along the outer margin of the orbit. Should it become necessary to divide an ocular muscle it can first be secured in the manner just described. This operation, carefully performed, may, in suitable cases, replace resection of the temporal wall of the orbit and produces much less deformity. Orbital cysts, dermoids, serous or blood cysts are treated in the same manner as growths, the dissection proceeding either through an incision along the orbital margin or, if conditions are favorable, through the conjunctiva, great care being taken to remove every particle of the cyst wall, often a difficult procedure. In some instances the cyst elaboration is so extensive that the eyeball cannot be saved.

Exostoses and **osteomas** growing from the wall of the orbit, or pushing their way into it from the ethmoidal or frontal sinus, may be reached by an ordinary dissection through an incision along the orbital

margin, with the usual precautions to avoid the pulley of the superior oblique, the tendon of the levator, and the lacrimal gland. After the body of the growth is fully exposed, it may be chiseled from its position in the ordinary manner or, if it is very dense and resisting, its base may be perforated several times with a drill suitably attached to a dental engine. It is next broken from its position with a stout pair of forceps, all rough spicules of bone carefully smoothed away, and the wound closed.

Resection of the Temporal Wall of the Orbit (*Krönlein's Operation*).—The operation, following Haab's directions, begins by dividing the soft parts with a curved incision (Fig. 332), which should be about 7 cm. in length in adults and 4 to 5 cm. in children, which commences above the supra-orbital margin and describes a gentle curve along the outer edge of the orbit to the upper edge of the zygoma, where it is bent backward and ends at the center of this structure. The center of this curved incision should bisect a horizontal line which connects the outer canthus with the

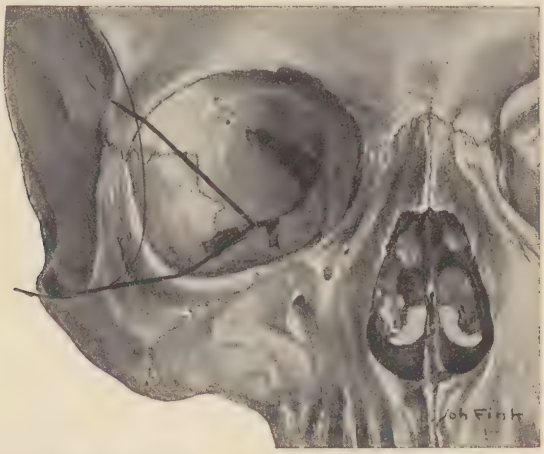


Fig. 332.—Skin incision (curved line) and bone incisions (heavy lines) in Krönlein's operation (Haab).

outer orbital margin, and here should be sufficiently deep to expose the opening of the orbit, while above and below only the skin and fascia and muscular layer are at first divided. Next, at a position corresponding to the central portion of this incision, a strong elevator is introduced, with which the periosteum is separated from the external orbital wall. The inferior orbital fissure is now localized, and, beginning at the anterior end of this fissure, the bony wall of the orbit is cut through with a chisel or with an electric saw, up and out to a point a little above the external angular process of the frontal bone, the line of incision being, for all practical purposes, along the suture between the great wing of the sphenoid and the malar bone, and outward and forward over the external surface of the malar bone in a line above the insertion of the zygomatic arch. Thus, a wedge-shaped piece of bone is formed, and with its muscular and cutaneous attachments is forced backward, giving free access to the orbit, which will be still partly covered with the periosteum. The latter must now be split from before backward and separated with retractors. This brings into view the external rectus muscle, and, if necessary, this may be divided near its tendinous insertion after the introduction of sutures, with which later the divided ends are united, or sometimes the muscle may be pushed aside and the dissection continued to the apex of the orbit. With suitable retractors the orbital fat

and ocular globe are pushed aside. After the exploration is complete, and this must sometimes be carried to the nasal side and the growth removed, the osteoplastic flap is replaced, the periosteum stitched with fine catgut sutures and the soft parts with silk. The question of drainage must be decided by the conditions remaining after operation. The usual full antiseptic dressing is applied, great care being taken that the lids cover the cornea, especially if the latter structure is anesthetic.

Various complications have occurred after this operation; for example, outward limitation of the eye owing to injury to the abducens, ptosis, sinking of the eyeball, and infection.

Indications.—Domela, Haab, and other writers have classified the indications for Krönlein's operation as follows: Retrobulbar cysts; tumors of the optic nerve and its sheath; neurofibroma of orbit (Parker); retrobulbar vascular growths, for example, cavernous angioma, lymphangioma, aneurysms, and varicose dilatations of the orbital veins; deep-seated foreign bodies in the orbit and exploration of the orbit in doubtful cases in order to establish a diagnosis. The operation has also been performed in deep orbital abscess, to open the sheath of the optic nerve in choked disk, and even for the removal of subretinal effusions. The two last indications are of doubtful value; certainly in choked disk a far better operation is a decompression-trephining.

Gifford recommends as a substitute for the Krönlein operation a definite resection of the outer wall of the orbit. He makes a horizontal incision $2\frac{1}{2}$ to 3 inches long, beginning $\frac{1}{4}$ inch from the outer commissure, care being taken not to open into the conjunctival sac. Next, the lips of the wound should be widely separated, and the periosteum pushed back from the outer side of the bone. Following this, with strong bone forceps the outer margin of the orbit and as much of the outer wall as desired is removed. Finally, the periosteum of the orbit is opened, and the operation terminates in the usual manner.

OPERATIONS FOR CATARACT

The following methods constitute the most important varieties of operation which are practised for the cure of cataract:

Extraction without iridectomy, so-called simple extraction; extraction with iridectomy, so-called combined extraction; extraction in the capsule; linear extraction; the needle operation, or discission; and the suction method. The old operation of *reclination, depressing,* or *couching,* as it has been variously called, by which the lens was forcibly thrust down into the vitreous, is rarely practised at the present time, although some surgeons have suggested that the operation is advisable in patients greatly enfeebled by age or other infirmities, if chronic conjunctivitis or dacryocystitis fails to yield to treatment, in lunatics, imbeciles, and others whose actions cannot be controlled, and particularly if one eye has been lost by intra-ocular hemorrhage.

1. **Needle Operation** (*Discission—Operation for Solution*).—By this operation the capsule of the lens is opened, the aqueous humor ad-

mitted to the lens matter, and absorption thus promoted. It is applicable to congenital and juvenile cataracts, and to some traumatic cataracts, and is rarely employed after the fifteenth year.

The instruments required are two cataract needles (lance-headed or knife-needle, according to the fancy of the operator), a stop speculum, and fixation forceps. The eye in this and all operations of similar character should be prepared in the manner described on page 651.

After the induction of general anesthesia in young children, or the use of cocain in older subjects, and full dilatation of the pupil, the operation is thus performed:

The lids being separated by the stop speculum, the surgeon fixes the eye with forceps, and enters the cataract needle through the cornea at its outer margin or at the limbus and carries it across to the center of the pupil, where the point is turned

Fig. 333.—Bowman's stop needle.

Fig. 334.—Knife-needle.

to the lens, and a laceration made in the capsule by depressing the handle of the instrument with a lever-like movement. Two cuts are made at right angles to each other, and the lens-matter may then be slightly broken up with the point of the needle. Care must be taken not to use so much force as to dislocate the lens, and not to lacerate too freely in the first operation, lest the lens substance, swelling up from contact with the aqueous humor, should produce injurious pressure on the iris and ciliary body. The operation usually has to be repeated at intervals, the second operation being performed after the swollen lens matter caused by the first incision has disappeared by absorption and the eye has become perfectly quiet.

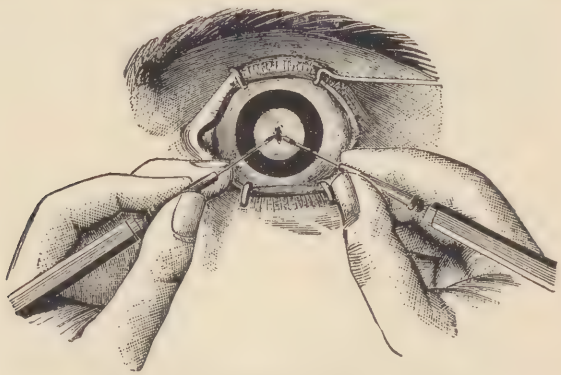


Fig. 335.—Discission with two needles.

At the second operation the needle may be used more freely, or two needles may be used in the manner shown in Fig. 335. The points enter the lens substance and the handles are approximated, thus making a decided separation in the remaining opaque matter. In order to prevent too deep entrance of the needle it is sometimes constructed with a shoulder (stop needle; see Fig. 333). In place of this procedure Ziegler's operation (see page 627) may be utilized. Instead of repeating the needlings, the first discission may be very free, with the understanding that in a few days it is to be followed by a linear extraction and a few weeks later by division of the capsule (C. F. Clark).

After-treatment.—The conjunctival sac should be irrigated with boric acid or physiologic salt solution, atropin freely instilled, and pupillary dilatation maintained during the entire treatment. Both eyes should be lightly bandaged.

Decided reaction, with hyperemia of the iris, pain, and ciliary congestion, indicates a more frequent use of atropin, dionin, and iced compresses. Great swelling of the lens matter, in addition to the symptoms of iritis, may give rise to a glaucomatous state. In these circumstances the lens matter which has escaped into the anterior chamber must be evacuated by a *linear extraction* or, what is practically the same thing, by a free paracentesis of the cornea. The *suction method* may also be employed in these circumstances. Some operators invariably extract the lens a few days after needling—a practice which certainly hastens the restoration of vision, but which is usually considered to be a procedure less safe than repeated discissions. C. F. Clark is not in accord with this belief and recommends linear extraction soon after a primary free needling.

For the *removal of the lens in high myopia* (see page 135) needling is employed. According to W. E. Lambert, the Fukala method, that is, needling the lens followed by a linear extraction, yields the best results in young subjects. If the patient is fifty or more years of age, and the lens is more or less cataractous, Lambert advises a preliminary iridectomy with subsequent extraction of the lens in the usual manner.

2. **The Suction Method.**—This operation is specially adapted for completely soft or fluid cataracts, and is also used, as has been stated, to remove lens matter which has been broken up by discission or by traumatism. It is performed as follows:

The pupil being dilated with atropin, the anterior capsule of the lens is freely lacerated with two needles. A small wound is made with a keratome passed obliquely through the cornea between its center and periphery. Through this opening and into the lens matter the "suction curet" is passed. This consists of a curet roofed in to within 2 mm. of its extremity, with a handle and a piece of India-rubber tubing furnished with a mouth-piece, which the operator applies to his lips and gently sucks out the lens matter into the syringe. This is Teale's method.

The same may be accomplished by using the syringe of Bowman, in which a sliding piston is worked by the hand. The point of the syringe must not penetrate too deeply, must be behind the lens matter which is to be removed, and must not be pushed back of the iris.

The after-treatment consists of rest, bandage, and the local use of atropin.

3. **Linear Extraction.**—This operation is designed for the removal of soft cataracts or those with a very small nucleus, and may be employed to remove lens matter after discission. Although any lens the substance of which is liquid enough to pass through a small corneal wound may be removed by this method, it is better, if possible, to restrict the operation to cases of soft cataract occurring in patients under thirty years of age, and to some varieties of traumatic cataract.

The following instruments are necessary: A keratome or lance-

shaped knife, fixation forceps, cystotome, curet, and stop speculum. The operation is as follows:

The surgeon fixes the eye with forceps, after the introduction of the stop speculum, wide dilatation of the pupil having previously been obtained, introduces the keratome about 1 mm. within the margin of the cornea, and makes a wound 5 mm. wide. The instrument is now carefully withdrawn, with a slight lateral motion to make the wound a little larger if necessary, and a sharp cystotome is introduced and the capsule of the lens is freely lacerated. The soft lens matter is now caused to extrude by counterpressure on the cornea with a metal spud, the outer lip of the corneal wound at the same time being depressed with a curet. This is a *simple linear extraction.*

The same manipulations may be performed, assisted by an iridectomy after the corneal section, a small segment of the iris being withdrawn with either hook or forceps and excised. Instead of using the cystotome to open the capsule of the lens, some operators do this with the keratome after making the incision in the cornea by causing the instrument to dip directly into the lens.

The after-treatment consists of bandage, atropin, and rest in bed until the eye is quiet.

Fig. 336. Fig. 337. Fig. 338. Fig. 339. Fig. 340.

Fig. 336.—Flap extraction.
Fig. 337.—Modified flap extraction (Knapp's section).
Fig. 338.—Modified peripheral linear incision.
Fig. 339.—Short 3-mm. flap with iridectomy.
Fig. 340.—Corneal incision.

4. **Extraction of Hard Cataract.**—It would be impracticable to indicate the numerous modifications which have been employed in this operation, than which, as the late Dr. Noyes has said, no surgical procedure has been more carefully studied and elaborated in every detail. Hence only a few well-recognized methods will be described.

(*a*) **Extraction without iridectomy,** often called *simple extraction.* The author is accustomed, following the directions of the late Dr. H. Knapp, to proceed as follows: The corneal section for full-sized cataracts comprises exactly the upper half of the cornea; for smaller, Morgagnian, and soft cataracts, somewhat less. A perfect section passes in its whole extent exactly through the transparent margin of the cornea, the knife (see Fig. 345) remaining in the same plane throughout, particular care being taken that in completing the section the blade of the knife is not turned forward nor backward. In many cases a small central con-

junctival flap is formed, which is an advantage. (For steps of operation, see pages 654–657.)

(*b*) **Extraction with iridectomy,** often called *combined extraction.* The peripheral linear extraction of von Graefe, by means of which the extreme periphery of the anterior chamber was opened by an incision 10 mm. long, through the sclerotic, 1 mm. external to the margin of the cornea and 2 mm. below the tangent of its summit, has been abandoned by almost all operators owing to its dangers—hemorrhage from the conjunctiva, loss of vitreous favored by the peripheral position of the wound and cyclitis, and consequently danger of sympathetic involvement of the other eye—and in its place one or other of the various so-called short-flap operations is performed.

A useful method is the following: A Graefe cataract knife is entered exactly at the corneoscleral junction at the outer extremity of a horizontal line which would pass 3 or 4 mm., according to the size of the cataract, below the summit of the cornea. Counterpuncture is made at a similar point directly opposite, and a flap is cut which embraces one-fourth or one-third of the cornea. A small conjunctival flap may be made or not. Iridectomy is performed. (For steps of operation, see pages 654–657.)

With the various *corneal incisions* which have from time to time been practised for the removal of cataract the author has no experience. Liebreich made an incision in the form of a curved section through the lower portion of the cornea, puncture and counterpuncture being effected in the sclerotic, while Lebrun caused the corneal flap to occupy the upper portion of the cornea and to be 3 mm. high, puncture and counterpuncture being made 2 mm. below the extremities of the transverse diameter of the cornea. In these operations iridectomy was usually omitted.

(*c*) **Extraction without capsulotomy** is performed by some surgeons—that is to say, the lens is delivered in its capsule. The operation was formerly chiefly employed to remove overripe cataracts and cataracts complicating high myopia with vitreous changes. Pagenstecher in these circumstances expelled the lens after an incision of about one-third of the corneal circumference and an upward iridectomy. The expulsion was accomplished either by pressure or with the aid of a spoon or loop. The chief danger of the operation is the risk of extensive loss of vitreous. The visual results are very good in successful cases. The author not infrequently has performed this operation, delivering the lens with a loupe after iridectomy through a section made in the corneoscleral junction.

Some ophthalmic surgeons of great experience in India and in this country believe that extraction in the capsule should be the operation of election. (For Colonel Smith's Method, see page 660.)

Preparation of the Patient and the Eye.—This should include a thorough examination of the patient, and the removal of the conditions already named (see page 406), which contraindicate the operation.

For some days previous to the operation, as H. Knapp insisted, the eye should be protected from anything which may produce congestion, and the patient should remain in the hospital, perfectly resting his eye and body, and frequently washing his face and the surfaces and margins of the eyelids with soap and water. This simple regimen will frequently change a congested and irritated conjunctiva into a pale and shining membrane. If there is any abnormal conjunctival discharge the instillation of a solution of argyrol (25 per cent.) is recommended by some surgeons, who also employ this drug in subsequent dressings of the operated eye (Callan). Bacteriologic examination should always be made, and if pathogenic organisms are present the operation should be postponed until they have been made to disappear by suitable treatment. Axenfeld recommends an injection of antipneumococcic serum prior to the performance of an operation in the presence of conjunctival pneumococci. During these days scrupulous attention should be given to the nasopharynx. The author, following a suggestion of J. A. Lippincott, of Pittsburgh, is accustomed to spray the nasopharynx three times daily with a solution of permanganate of potassium (1 : 5000), with gratifying results. Dr. J. A. White recommends that the conjunctival sac shall be filled with bichlorid-vaselin (1 : 3000) on the night prior to the operation, where it remains until the next day.

The preparation of the skin of the region of operation, and particularly the ciliary margins, has been described on page 593. These preparations should be made at least two hours before the operation, and the eyes should be covered with squares of lint soaked in a solution of bichlorid of mercury (1 : 5000), held in place with a gauze roller. Just preceding the operation, the preparatory bandage having been removed, the ciliary margins may again be washed with soap and water, followed by bichlorid of mercury (1 : 5000), with the same precautions previously described (see page 593). Next, the conjunctival culdesac should be flushed with a tepid solution of boric acid applied with some force, or with a sterile physiologic salt solution. During these irrigations pressure should be made over the lacrimal sac in order to be sure that no deleterious secretion is contained within it. The canaliculi and lacrimal canal may be irrigated with a boric acid or saline solution introduced by means of an Anel syringe. The lids are next everted, the tarsal conjunctiva and the region of the inner canthus wiped with a pledget of cotton moistened in the boric acid solution. The cornea should be anesthetized with three instillations of a sterile 4 per cent. solution of cocain, applied at intervals of five minutes, and the eye carefully closed and covered with the antiseptic pad after each instillation. In place of cocain some surgeons prefer holocain in 2 per cent. solution or a mixture of holocain and cocain. Just before the knife is entered the surface of the cornea should be carefully wiped with a pledget of cotton soaked in boric acid solution. This same method of preparing an eye should be practised not only in cataract extraction and discission, but also prior to all operations requiring corneal incision—for example, iridectomy, iridotomy, etc. With

injection of physiologic salt solution prior to extraction to deepen an abnormally shallow anterior chamber the author has had no experience. He has not deemed its wise to flush the conjunctival sac vigorously with strong solutions (1 : 2000 or 3000) of bichlorid of mercury, but this practice is commended by surgeons of great experience, for example, Col. Henry Smith. Placing a preparatory dressing over the eyes during the night preceding the operation the author is satisfied is an unwise procedure.

Fig. 341.—Lid-elevator.

Position of the Patient.—The patient during the operation should lie, according to the custom of the operator, upon an operating chair or table. If, as is sometimes advisable, the operation is performed while the patient reclines in bed, the head should rest on a moderately hard cushion or pillow, covered with a sterile sheet, another pillow at the same time supporting the shoulders,

Fig. 342.—Metal spoon.

Fig. 343.—Wire loop.

Fig. 344.—Cystotome.

Fig. 345.—Cataract knife.

Fig. 346.—Capsule forceps.

so that the position is as little strained as possible. In all circumstances the face must be turned so that uniform daylight falls upon it, or the area of operation should be illuminated with a suitable electric lamp, the model of Ziegler being most satisfactory.

Instruments, Solutions, and Dressings.—The instruments required are the following: A stop speculum, a lid-elevator, a large strabismus hook, a spatula, a wire loop, a spoon, an olive-tipped probe, a curet, a cystotome, capsule forceps, a pair of scissors, iris forceps, iris scissors, and the cataract knife.

The following lotions and dressings should be at hand: Atropin drops, 4 grains (0.26 gm.) to the ounce (30 c.c.); eserin drops, $\frac{1}{2}$ grain

(0.0324 gm.) to the ounce (30 c.c.); cocain solution (4 per cent.); saturated solution of boric acid; two solutions of bichlorid of mercury (1 : 5000 and 10,000), and boiled distilled water containing 0.5 per cent. of chlorid of sodium. Suitable bulb syringes and an irrigating apparatus, for example, Lippincott's, should be ready.

For the purpose of dressings the following may be needed: Several rollers, 2 inches wide and 5 yards long, made of sterilized gauze, and sterilized oval pads of lint and absorbent cotton. A useful bandage is composed of a broad band of knitted material which is tied with four tapes, which pass above and below each ear.

Everything being in readiness, the operation may be performed as follows:

The surgeon, if he is ambidextrous, may stand behind the patient, no matter which eye is to be operated upon; if he is not, he should take this position for the right eye only, standing at the patient's side and in front for an operation on the left eye. Again, if the surgeon is ambidextrous, he may stand in front and at the patient's right side for an operation upon the right eye, and at the patient's left side and in front for an operation on the left eye.

The speculum having been inserted, the surgeon steadies the eyeball and draws it downward with the fixation forceps (it is supposed that the section is being made upward) by taking firm hold of a fold of conjunctiva below the inferior border of the cornea (some surgeons prefer fixation at the inner side, over the internal rectus tendon), enters a Graefe cataract knife exactly at the corneoscleral junction, as before described, at the outer extremity of a horizontal line which would pass 3 or 4 mm., according to the size of the cataract, below the summit of the cornea, passes across the anterior chamber to a corresponding point upon the opposite side, and makes the counterpuncture. The knife is pushed steadily onward as far as possible, with an upward tendency, and the incision is completed by a free cutting, not a sawing or dragging movement, keeping the knife in the same plane throughout, and not turning its edge at the completion of the section either forward or backward. This maneuver will create a small conjunctival flap. If this is not desired, when the summit of the cornea is reached the knife must be turned a little forward before the completion of the flap. It is the practice of some surgeons to remove the speculum as soon as the section is completed; other operators prefer not to use a speculum, but to separate the lids with their fingers or with a lid-elevator or a Smith's or Fisher's hook held by an assistant. This completes the *first stage* (Fig. 347).

In the *second stage*, or the stage of iridectomy, the fixation forceps are intrusted to the assistant (trained to hand the instruments in their proper order), who gently draws the eyeball downward, while the operator takes in his left hand the iris forceps and in his right the iris scissors. If the iris is already protruding in the wound, a small portion of it may be seized and snipped off with a single cut close to the border of the cornea. If not, the blades of the instrument must be introduced in the manner described under Iridectomy, and the pupillary border of the iris seized, the tissue drawn out and toward the cornea, and cut off close to the cornea. It is not necessary to make a large coloboma. If the patient is to be trusted, it is not necessary that the assistant shall draw the eyeball downward while iridectomy is being performed. The patient may simply be directed to look downward while the surgeon proceeds to remove a small portion of the iris in the manner already described. The pillars of the coloboma should now be carefully smoothed out with a delicate spatula. This completes the *second stage* (see Fig. 306).

In the *third stage*, or the stage of capsulotomy, the operator takes in one hand the fixation forceps and gently steadies the eyeball, while with the other he introduces the cystotome, held flatwise during its insertion, passes it to the bottom of the coloboma, and then turns its cutting-edge toward the capsule. From this point a vertical incision is traced until the upper portion of the coloboma is reached, where a trans-

verse cut is made. Great care should be taken to cut, and not to tear, and the whole maneuver should be accomplished without undue pressure lest the lens be dislocated. Other methods of opening the capsule are the following: Two cuts inclined to each other are made like the limbs of the inverted letter ∀, together with a transverse cut at the periphery; or, as was recommended by H. Knapp, the capsule may be opened in its extreme periphery, with the understanding that later on the necessity for the operation for after-cataract will arise. Some surgeons open the capsule with *capsule forceps*, as a rule, and this method is especially recommended by E. Treacher Collins; the author prefers to use this instrument only in cases in which the anterior surface of the capsule is thickened. In withdrawing the cystotome the operator should again turn it flatwise, and be careful not to drag any tags of capsule into the wound. This completes the *third stage*.

In the *fourth stage*, or that of delivery of the cataract, the operator draws the eye slightly downward, or, if he has a docile patient, causes him to look downward, while the assistant raises the speculum so that its blades shall not press upon the eyeball and yet shall hold the lids away from the eye or lifts the upper lid with a Smith's hook or with an elevator, at the same time drawing away, with his thumb, the lower lid from contact with the globe. The back of a curet or the convex surface of the

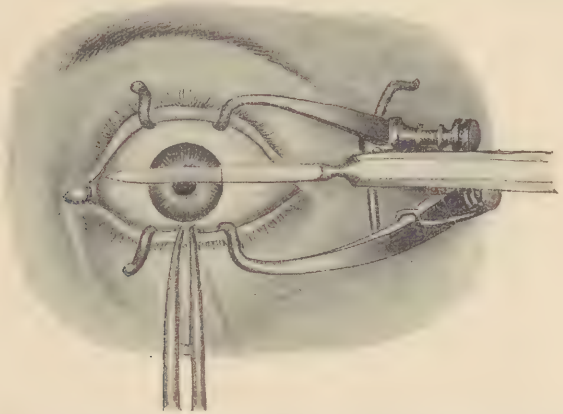


Fig. 347.—The incision in cataract extraction. Puncture and counterpuncture have been made. The section will pass in its whole extent exactly through the transparent margin of the cornea, the knife remaining in the same plane throughout.

metal spoon is now laid against the inferior portion of the cornea, and firm but at the same time gentle pressure is made, causing the upper margin of the lens to appear in the wound. The pressure is exercised with an upward motion to coax out the cataract, but is relaxed as soon as the major portion has been expelled, in order that no undue tension be put upon the zonula. As the cataract slips through the wound the spoon is made to follow it, catch it, and lift it out with a little sweeping motion which may at the same time remove any small fragments of the cortex which have broken off and lie at the margins of the incision. The speculum or lid-elevator is then removed. This completes the *fourth stage* (Fig. 348).

In the *fifth stage*, or that which is now called the "toilet of the wound," after the eye has been allowed to remain closed for a few moments the operator cautiously inspects the wound, after raising the upper lid with his fingers, or preferably with a lid-elevator, while the patient looks downward. In this inspection he should ascertain whether the pupil is clear or whether any cortical remnants are present or tags of capsule lie between the lips of the incision. If cortical matter remains, it should be removed as follows: The eye being turned downward, the operator makes a gentle rubbing movement in an upward direction on the cornea with the convex surface of a horn spoon, great care being taken not to press too hard lest vitreous escape. By

rubbing gently in a circular manner the cortical particles will gather in the upper part of the wound, and then, while the slight pressure continues, the lips of the wound may be gently separated with the metal spatula and the expulsion of the cortical remnants effected. Blood-clot, the result of hemorrhage from the iris, may be expelled in like manner. While these manipulations are being made, the author is accustomed to flood the surface of the eye and lips of the wound with a physiologic salt solution. After they are completed, a final inspection is made, and in order to be sure that no tag of capsule remains in the wound, or that no portion of the conjunctival flap has been caught between its lips, the olive-pointed probe is gently passed from one end of the incision to the other.

Some surgeons advocate *irrigation of the anterior chamber*, which, as has already been stated, is used also in the operation of unripe cataract. In this maneuver the tip of a specially devised syringe is introduced between the lips of the wound, and the irrigating liquid injected, which causes blood-clot or cortical matter to be washed out. If irrigation is employed, two cautions are necessary: (*a*) No strong antiseptic solution should be used, certainly never bichlorid of mercury, which is liable to produce indelible staining of the cornea. The irrigating fluid should be boiled distilled water containing 0.5 per cent. of the chlorid of sodium. (*b*) In passing the liquid from the syringe into the anterior chamber, the direction of the flow should be over

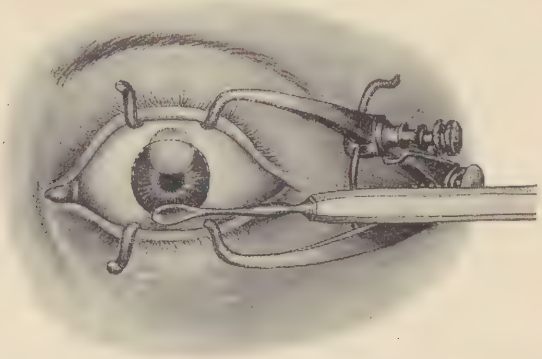


Fig. 348.—The delivery of the lens; the lens is presenting in the wound (capsulotomy has been performed).

the wound from within outward, and not the reverse, lest particles of blood and cortex be driven inward.

A general inspection of the conjunctival sac may now be made; sometimes a little blood-clot or a cilium may be present. In wiping away any clots, delicate pieces of sterilized gauze are very suitable, or the clots may be picked up with the iris forceps. If all these manipulations have been successfully performed, the conjunctival culdesac will be free from foreign matters, the edges of the wound nicely coapted, the pillars of the coloboma as straight as possible, and the angles not caught in the margins of the wound, the pupil black, and the patient readily able to count fingers. This completes the *fifth stage*.

If the operator intends to perform *extraction without iridectomy*, the following additional directions will be found useful. As the author is accustomed to perform the operation according to the late Dr. Knapp's rules, the advice of this surgeon is quoted:

After performing the section according to the method already given (see page 654, also Fig. 347), the expulsion of the lens is effected by pressing the lower part of the cornea with a Daviel spoon directly toward the center of the globe. When the lens presents in the gaping section, its exit is aided by slight strokes with the spoon

on the outer surface of the cornea. If the sphincter proves to be rigid, it may be drawn backward with a wire loop or with a special iris retractor, and usually it is safer to remove both fixation forceps and speculum immediately after the corneal section and during the process of expelling the lens, or the speculum may be raised in the manner already described. If desirable, the upper lid may be elevated in these circumstances with a large strabismus hook or with a lid-elevator. The pupillary space should be cleared by pressing on the cornea with the edge of the lower lid—care being taken that it does not come in contact with the lips of the wound—or, better, with the convex surface of a polished spoon. The cortical remnants are wiped away with a probe-pointed curet. During this operation the lips of the wound may be flooded with the boric acid or sterilized salt solution (Knapp used a 1 : 10,000 solution of corrosive sublimate).

The concluding steps of the operation are described in Knapp's own words: "The conjunctival flap is smoothed out by introducing the end of a polished grooved spatula, previously sterilized, into the anterior chamber, and passing it through the wound from one end to the other, stroking from within outward, in order to remove particles of lens, redress a curved-in flap, and carefully adjust the edges of the wound. This is, however, not done before the iris has spontaneously or artificially recovered its natural position. Should the corneal section be too peripheric, the best thing is to make a small iridectomy at once, for peripheric (Graefe's) sections commonly lead to large and harmful prolapses. If the iris does not spontaneously resume its position, frequently it does so when the lower part of the cornea is pressed upon with the edge of the lid. This paradoxic phenomenon may thus be explained: The iris being pinched in the tightly closing wound, pressure on the part of the cornea raises the flap and disengages the iris, which then, by its natural elasticity and contraction of the sphinter pupillæ, can resume its natural position. If this procedure fails, the iris should be pushed back with a spatula into the anterior chamber. When the periphery of the iris remains folded in the sinus of the anterior chamber, it is smoothed out with the olive-tipped point of a probe introduced into the iris angle behind the opaque corneal margin."

Fig. 349.—Ring's ocular mask.

The final stage of all cataract operations is the application of the dressing. Much difference of opinion exists upon this subject. Some operators simply close the lids with a strip of isinglass plaster, while others place upon them an elaborate bandage.

The author is accustomed to use the following dressing: An oval piece of soft lint soaked in a solution of boric acid is laid upon each closed lid, the margins of which have previously been liberally smeared with bichlorid-vaselin (White's ointment, see page 652); over this is placed a similarly shaped piece of sterilized cotton, large enough to be flush with the eyebrow and lower margin of the orbit, and is held in place with three narrow strips of surgeon's isinglass plaster, passed from the inferior edge of the orbit to a point above the brow. Over this a very light, single piece of knitted bandage is tied by means of four tapes which pass above and below the ears. The entire dressing is usually covered with the mask devised by Dr. Frank Ring, of New York (Fig. 349). The patient is put to bed in a comfortable position in a slightly darkened room, although with the aid of the mask the latter precaution is unnecessary, and the patient may remain in the open ward of the hospital or in an ordinary room without danger. If the caruncles are tumid, or if there has been any suspicious secretion from the lacrimal sac, the author is accustomed to fill the inner canthus with dry sterile iodoform powder, which forms a small cake and prevents access of infection to the wound.

After-treatment.—For the first few hours, the effects of the cocain having passed away, there are some smarting and burning, but severe pain should not occur. If at the end of twenty-four hours after a combined extraction there has been no discomfort, no headache, and nothing to indicate that any anomaly in the course of healing is going on, the dressings need not be removed; but if they have become disarranged or the patient has been uncomfortable, they should be taken off and the lids inspected. A little staining of the strip of lint is of no consequence, and if the eyelids are not swollen and there is no discharge and the delicate veins in the skin of the lids show no distention, the eyelids need not be opened and the dressing may be reapplied; or the lower lid may be gently drawn downward so as to permit the escape of tears which may have accumulated in the conjunctival culdesac, or to liberate the eyelashes if they have become inverted. At the end of forty-eight or seventy-two hours the wound may be inspected by candle-light, a drop of sterile atropin solution instilled, and each succeeding day the usual dressing reapplied; at the end of three days the dressing may be removed from the unoperated eye, and at the end of a week or even earlier the patient needs only a shade and dark glasses. Although some operators do not require cataract patients to go to bed at all, it seems to the author that it is safer to keep them in bed for two or three days. The recumbent posture too long maintained may lead to hypostatic congestion of the lungs. Sometimes elderly patients are very uncomfortable when confined to bed and become slightly delirious; in these circumstances they may be allowed to rest in an easy chair. For a few days liquid food, or at least food which does not require much chewing, should be given; after this the ordinary diet suited to the patient is permissible.

Some surgeons prefer the "open method" of managing eyes after cataract extraction—*i. e.*, no occlusive dressing is applied, but the eye is protected with spectacles made of wire-gauze or similar material. With this procedure the author has no experience.

If the operation has been an extraction without iridectomy, it is proper to inspect the eye at the first dressing, usually at the end of twenty-four hours, in order to ascertain whether there has been any prolapse of the iris. Should this accident have occurred, the treatment must be pursued according to the directions given elsewhere. If the iris is in place and the pupil circular, although it is proper to change the dressings once in twenty-four hours, it is unnecessary to inspect the line of incision. All that is required is to draw down the lower lid and permit the escape of any accumulated tears. As soon as the wound is closed, a drop of a sterile atropin solution may be instilled and this instillation repeated at subsequent dressings.

Accidents.—The following accidents may occur during the performance of a cataract extraction:

1. The knife may be introduced with the cutting edge turned in the wrong direction. If this somewhat inexcusable mistake should occur, the knife must be withdrawn and properly inserted. If this cannot

be done, owing to the escape of the aqueous, postponement of the operation until the anterior chamber has refilled is necessary.

2. The conjunctiva in the neighborhood of the counterpuncture may become distended with aqueous humor. This produces an elevation resembling a bleb. The section should be completed as if the accident had not happened.

3. The iris may fall before the knife. The incision should be completed in the ordinary way. An irregular coloboma will result, which may be remedied by seizing the jagged edges with the iris forceps and trimming them with the scissors.

4. Free hemorrhage may occur if a conjunctival flap is made or in performing the iridectomy. Under pressure the bleeding will sometimes cease, and the operator should then endeavor to get rid of the blood in the manner already described. If success does not follow the maneuver, the cystotome must be introduced, even though everything is obscured by the blood, the capsule lacerated, and the lens expelled. During its expulsion sufficient blood will often come away to clear the pupillary space.

5. The wound may be too small. This is a very unfortunate occurrence and can be remedied by enlarging the incision, which is best done with a small pair of probe-pointed scissors.

6. Undue pressure of the cystotome may cause the lens to be partially or completely dislocated. If the dislocation is partial, the eyes should be closed and gentle pressure should be made with a bandage; the lens probably will right itself and can be delivered. If the dislocation is complete and the lens slips back into the vitreous, it must be removed by means of the scoop or wire loop or by pressure (see page 413).

7. The vitreous may escape before or after the expulsion of the lens. If before the expulsion of the lens, the operator should at once remove the cataract with the wire loop, which is gently inserted behind the lens. At the same time all pressure upon the eye must be removed. If vitreous escapes after the lens has been extracted, the wound should be cleared of protruding vitreous as gently and rapidly as possible and a bandage applied. The patient should be required to look upward, as a downward position of the eye favors the prolapse of the vitreous. Although escape of vitreous is an undesirable accident, its consequences are not always serious and good visual results may be obtained. If the escape of vitreous has been great, particularly if the vitreous is thin and there is tendency for the eyeball to collapse, a tepid sterile physiologic salt solution should be injected into the vitreous chamber until the globe assumes its proper contour, as has been recommended by J. A. Andrews and Herman Knapp.

8. Occasionally the corneal flap is everted because it has been caught by the margin of the lid, owing to a sudden movement of the patient. It must be replaced and a bandage quickly applied. Sometimes immediately at the conclusion of the section, or directly after the delivery of the lens, especially in old and feeble subjects, there is great collapse of

the cornea, which, instead of keeping its proper curve, looks like a wrinkled membrane. In these circumstances the anterior chamber should be filled with physiologic salt solution, which will not only aid in making proper coaptation of the lips of the wound, but will prevent the sucking in of the conjunctival juices which might lead to infection.

9. Capsulotomy may not have been sufficient and pressure upon the inferior half of the cornea fails to cause the lens to present. In such a case the cystotome must be reintroduced and the laceration enlarged, or if the obstruction is due to the presence of a tenacious center in the capsule, this may be removed with capsule forceps.

Within the last few years Colonel Henry Smith's method of extraction of cataract in the capsule (radical operation for cataract, according to Vail) has attracted much attention.

Smith's Operation for the Extraction of Cataract (*Indian Method of the Extraction of Cataract in the Capsule*).—The operation is performed as follows:[1]

After a spring speculum has been inserted, the eye being fixed with special forceps, a Graefe knife is entered at the corneoscleral junction and counterpuncture made in the corneoscleral junction of the opposite side, so that the incision when completed shall include half or nearly half of the circumference of the cornea. The knife is driven through to the heel, the handle being lowered as it passes onward and the point elevated. If the manipulation is a proper one, after the counterpuncture is made the knife cuts as it goes through the tissues, and the incision is completed with one thrust, the incision ending in the cornea in such a manner that the edges of the corneal wound are cut as nearly as possible at right angles to the surface. An iridectomy may or may not be done, but it is advisable, for the beginner at least, to make this part of the operative procedure.

The speculum is next removed, and the eyelid and brow are held away from the eye by means of a large blunt hook and the assistant's fingers (Fig. 350). The patient is required to look steadily upward. In the case of an immature lens or of a hard cataract the operator next presses back with the point of a strabismus hook toward the optic nerve, the point of the hook being placed over the lower third of the cornea. This pressure must be steady, and the point of the hook should not be removed until the upper edge of the lens tilts forward. The moment the lens is seen to be dislocated, the pressure through the point of the hook is gradually turned more and more toward the wound, pressure during all of this time being maintained, so as to keep the lens up to the sclerotic margin, the pressure with the hook becoming gradually lighter and lighter and the hook gradually sliding under the lens until the cornea is folded beneath it. At this stage the lens is delivered (Fig. 351).

If the tension of the eye is low, the emerging lens should be followed with a spatula as well as with a hook, and sometimes, in these circumstances, a light counterpressure with a spatula above the wound is indicated.

In the case of intumescent lenses and Morgagnian cataract, the capsules of which are extremely delicate and liable to burst, pressure with the point of the strabismus hook is made sufficiently deep over the lower border of the lens to cause it to dislocate below and turn a half somersault, the pressure and traction always being made over the zonula. As soon as the lens turns up into the wound the operator ceases to make traction toward the patient's feet and directs the pressure backward, next backward and upward, and finally more and more toward the wound, folding the cornea beneath the lens until it falls outside of the cornea. As the capsule has not yet been dislocated from the zonula in the neighborhood of the wound, the hollow of the curve of the strabismus hook should be made to sweep along between the

[1] This description, condensed and slightly modified, is taken from the late Dr. D. W. Greene's quotation of Colonel Smith's own account of his operation.

lens and the wound to complete this detachment. It is advisable in these circumstances to keep up sufficient tension on the eyeball with the spatula placed on the cornea in order to prevent the lens from slipping back into the eye.

Hypermature cataract is difficult to dislocate, and the operator should proceed with the strabismus hook as in the case of immature and hard cataract, but a spatula in his left hand should be in position at the margin of the wound to drop behind the lens the instant its edge appears. As it is dropped almost straight down into the eye, the back of the spatula must be placed against the sclerotic margin of the wound and the lens pressed against it with the hook from the outside. The lens, therefore, slides along the inclined plane of the spatula.

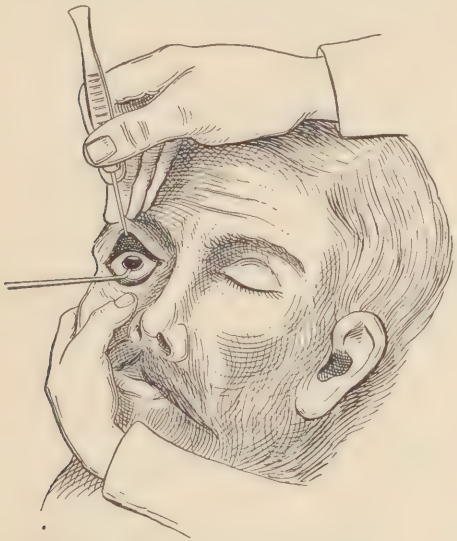


Fig. 350.—Smith's operation for cataract: Spectator's view, while the lids are being held by the assistant and the hook for expelling the lens is applied. Notice the gable-like space under the upper lid above the eyeball. The operator may look obliquely under the upper lid in this field and get a good exposure of the entire upper *culde-sac*. The assistant can shift this exposure to the right or left to enable the operator to have an unobstructed view of the lens and wound; also to give him access to the angles of the wound and the summit when replacing the cut sides of the iris, and the apron of the iris that adheres to the scleral wound after delivery. (Description and illustration by Vail.)

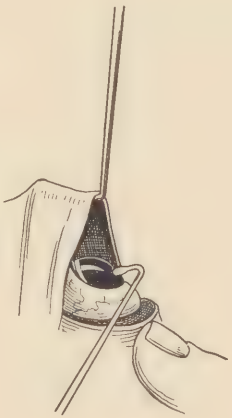


Fig. 351.—Smith's operation for cataract: Shows where the bulbous end of the lens hook is applied in the act of pressing to expel the lens. The pressure is made "straight back toward the optic nerve," not quickly or plungingly, but with intelligent and bold onward pressure, never varying the direction of the pressure until either the lens breaks from its moorings above or below and shows a disposition to be delivered, in which case the exit is favored by shifting the pressure, or the lens stubbornly refuses to yield and advance because of tough ligaments or small size of the incision. (Description and illustration by Vail.)

Great care should be taken to return the iris to its proper position, whether or not an iridectomy has been made, and with a suitable repositor the operator should release any part of the iris from the sclerotic or from the angles of the wound. If this is carefully done, the eye need not be dressed until the expiration of the eighth day, and many of the complications which have been described may be attributed to meddlesome dressings and meddlesome inspections. If vitreous escapes, it should be snipped off with the scissors in the usual manner.

According to Colonel Smith, cataracts occurring in children and young persons are not suited to this operation, because it is practically

impossible to dislocate their lenses. The operation seems to be valuable in unripe cataract, and in those forms of cataract which are immature and which require long periods of time for their complete opacification. Much difference of opinion exists as to the value of this operation and its permanent place in surgical practice. In our own country the operation was particularly recommended by the late D. W. Greene. Derrick T. Vail, A. Timberman, and especially W. A. Fisher (who has suggested useful modifications and who has designed an admirable lid elevator), who have had large opportunities of learning the technic in Colonel Smith's clinic in India, are enthusiastic advocates of this procedure. It has also received the commendation of Robert Sattler and other American surgeons.

One of the objections to the Indian operation is that the percentage of loss of vitreous is much higher than in the ordinary extractions of cataract. Colonel Smith has reported his vitreous loss to be 8 per cent., while Vail, in a series of uncomplicated cataracts, did not exceed 2 per cent. of vitreous loss, and therefore he believes, to use his own expression, that vitreous escape is not the argument to be used against Smith's technic. W. A. Fisher, although admitting the disadvantage of frequent vitreous loss, believes this can be overcome by careful technic and that Smith's operation should be the procedure of choice. Naturally, the ordinary accidents of cataract extraction are liable to occur, but apparently in Smith's Indian service they are not greater than those which occur in ordinary extraction. It is the author's impression, based on a very limited experience and upon his observation of certain operators who are familiar with the Smith technic, that while this operation will retain a place in ophthalmic surgery, especially in the extraction of unripe cataracts, it is not likely to drive from the field those procedures which have for years been firmly and favorably established.

Several operations for the extraction of cataract in the capsule after subluxation of the lens with capsule forceps have been devised. Stanculeanu's operation, which has been commended by Simpson and Howard Hansell, in its main features is as follows:

> After an incision in the limbus somewhat larger than the usual one, a sickle-shaped forceps is entered into the anterior chamber, and a fold of capsule is grasped within its branches. By horizontal, upward, and downward movements the zonula is ruptured. When the operator feels that the lens has been dislocated, the forceps is withdrawn, and the lens is expressed in the usual manner with the aid of two spatulas.

Arnold Knapp's method is as follows:

> "After the usual preparation and the instillation of 1 drop of atropin, under holocain anesthesia, the Koster speculum is introduced and left in place until the operation is completed, unless there is danger of or actual prolapse of vitreous. An assistant is necessary only in the presence of complications. The section must be large and should be just short of half the corneal circumference with a conjunctival flap. After the iridectomy, the capsule forceps is introduced to a point below the center of the pupil, the branches are then allowed to separate broadly,

and a distinct knuckle of capsule is grasped. The grasp should not be too tight lest the capsule be torn, but sufficiently firm to exert traction on the periphery of the lens capsule. The closed branches of the forceps are gently moved from side to side, up and down or rotated, and the capsule can be seen to follow in the various directions. When the dislocation has succeeded, a part of the margin of the cataract in the capsule appears free in the pupillary space. The portion dislocated is usually below, generally slightly to one side or the other, with the upper attachment unruptured. The forceps is then released and withdrawn. Pressure is exerted straight back on the lower part of the cornea with Smith's hook, and the cataract can be seen to turn a somersault; it "tumbles," in other words, as Smith calls it, and is delivered feet first. When the entire lens has been delivered, it will be found adherent above, where it is finally separated by a lateral stroking motion. In some cases the head presents first; the delivery is then slower, and counterpressure must be applied at the scleral margin. The iris columns are then carefully replaced. The coloboma should ultimately not appear any different from that after an ordinary extraction."

To prevent iris and vitreous prolapse a number of operators have placed, prior to the extraction, a suture, *Kalt's corneal suture* in this regard being well and favorably known. The point of a sharp needle, armed with fine linen thread, is passed into and out of the *substantia propria* on one side of the proposed section and next obliquely through the opposite border of the limbus and through the scleral tissue. The loop thus formed is drawn aside, the incision completed, and after the delivery of the lens the threads are tied in a single knot. *Sliding conjunctival flaps*, which cover the line of incision, for example, the Van Lint flap, recommended in certain cases by L. Webster Fox, have also been used to prevent iris prolapse and infection. A number of operations whereby a *subconjunctival extraction of cataract* is performed have been devised, for example, by Dimmer, and in this country by Frank Todd.

Anomalies in the Healing Process after Cataract Extractions.—*Pain.*—Should pain occur and not be due to the circumstances already mentioned, but become violent in character, either in the earlier stages after the operation or some days afterward, one of three things may be apprehended: intra-ocular hemorrhage, suppuration of the wound, or iritis.

Expulsive Intra-ocular Hemorrhage.—Usually, soon after the operation has been completed, the patient complains of very severe pain, or vomiting may occur and the dressings begin to be stained with blood. On removal of the bandage a clot of blood will be found protruding through the palpebral fissure, and on raising the lid the anterior chamber is seen to be full of blood and the corneal wound gaping widely. As soon as the symptoms of this accident are manifest, the patient should be placed in an upright position and a hypodermic injection of morphin administered. The blood should be carefully removed, the conjunctival sac washed out with a bichlorid solution (1 : 8000), and a full antiseptic dressing applied. The dressings should be changed once or twice daily. In this way it may be possible to avert suppuration, even though the eye remains blind. If the hemorrhage should continue and the pain become intense, enucleation is necessary.

Suppuration of the Wound.—According to E. T. Collins, purulent infection is more common in old people than in young, and the tendency is greater between sixty and seventy than between seventy and eighty, though it is certainly greater between eighty and ninety than between sixty and seventy. It may be caused by lacrimal complication, inflammation of the upper respiratory tracts, conjunctivitis, blepharitis, by infection introduced at the time of the operation, or during the first dressing of the eye, for example, a non-sterile atropin solution. Suppuration commences on the first, second, or third day, more rarely on or after the fifth day, but sometimes as late as the thirteenth day.

The symptoms are pain, swelling of the lids, chemosis of the conjunctiva with undue secretion, haziness of the cornea, turbidity of the aqueous, and the formation of a slough along the margins of the wound.

Two terminations are possible: The suppurative process may be limited, so that at the end of the inflammation the pupil is closed and

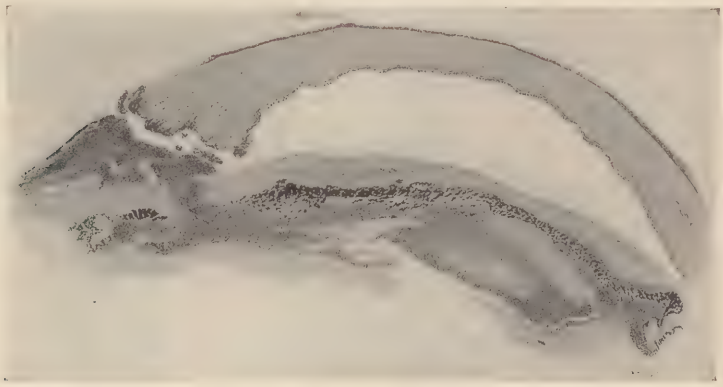


Fig. 352.—Section of an eye with postoperative infection which began on the ninth day after spontaneous reopening of the wound. Notice the dense infiltration of the wound edges and that the lens capsule has been caught in this material between the lips of the wound.

the iris drawn upward, or the entire globe may participate in a general destructive inflammation (purulent panophthalmitis).

If the suppuration is limited to the margin of the wound, prompt treatment may be of avail. The conjunctival sac should be carefully disinfected, the lips of the wound gently parted after removal of the slough and irrigated with a bichlorid solution, and the whole line of incision freely cauterized with the actual cautery or with liquid carbolic acid. In other words, the treatment is practically that which has been advised for a sloughing ulcer. At each subsequent dressing the lips of the wound should be parted with a probe, and the anterior chamber drained. Argyrol solution (25 per cent.) may be instilled into the conjunctival sac, and its introduction into the anterior chamber has been advised. Hansell recommends the injection into the anterior chamber of a few drops of a solution of bichlorid of mercury (1 : 1000). Ziegler advises the constant application of ice and the application of formalin to

the wound and its injection into the anterior chamber. Subconjunctival injections of bichlorid of mercury or cyanid of mercury have been recommended in these circumstances, and the introduction of iodoform into the anterior chamber has been suggested, and successes have been reported. In recent times postoperative suppuration has been treated with *vaccines;* for example, the injection of Wright's *antistaphylococcic vaccine* (Morax) and the administration of an emulsion of staphylococci by the mouth, the dose being gradually increased from 100,000,000 to 700,000,000 (Grimsdale). A bacterin prepared from the micro-organism which is active has in more than one instance proved highly successful, and should certainly be given full trial. The bacterin treatment may be combined with the internal administration of urotropin. Should the infecting micro-organism prove to be the pneumococcus, *ethylhydrocuprein* may be tried (see page 256).

If the infection manifests itself in the form of a *ring abscess*, treatment is usually unavailing, and the eye passes into a state of panophthalmitis and requires the treatment for that condition which has already been detailed. Suppuration, instead of beginning in the cornea, may sometimes commence in the iris and even in the vitreous, and the process go on to a rapid destructive panophthalmitis. It is a clinical fact that if one eye has been lost on account of postoperative suppuration, the other eye, if submitted to operation, is in grave danger of meeting a similar fate.

Iritis and Iridocyclitis.—It is not uncommon for attachments to form between the capsule of the lens and the margin of the pupil or of the coloboma. These may be regarded as non-inflammatory synechiæ and are not of serious consequence. Iritis itself, with the usual symptoms of this condition, generally sets in about the fifth day, but may be delayed to the tenth day. It may be caused by an imperfect toilet of the wound, with the retention of pieces of cortex, and sometimes by too early exposure of the eye, but usually should be regarded as a manifestation of infection. If the ciliary body becomes involved and an *iridocyclitis* is set up, the gravity of the situation increases and the process may terminate in distortion and closure of the pupil, with exuded lymph. Iridocyclitis may last for weeks, the inflammatory symptoms varying in their intensity, but finally the iris becomes dull and discolored and there is grave danger of sympathetic trouble in the opposite eye. Indeed, sympathetic ophthalmitis in these circumstances has been reported a number of times. *Late cyclitis*—that is, an inflammation occurring after the first week, is characterized by deep-seated circumcorneal injection, thickening and opacity of the capsule, and posterior synechiæ. Under treatment the symptoms may subside or secondary glaucoma may develop.

The *treatment* of these conditions in general terms should include bleeding from the temple by means of leeches, the free use of atropin, dionin, holocain, hot fomentations (usually recommended, but iced packs are preferred by some surgeons, and generally are more satisfactory than heat), the internal administration of full does of salicylate

of sodium, and in most circumstances mercury and iodid of potassium. A number of observers have obtained good results from the administration of neosalvarsan. If the process closes the pupil, after the eye becomes quiet iridectomy, iridotomy, or iridocystectomy may be required.

A remarkable condition to which H. Knapp called special attention is the formation of a *spongy* or *gelatinous exudation* in the anterior chamber, associated at first with considerable pain, congestion of the conjunctiva, and edema of the margins of the lid. The manifestations are those of spongy iritis without an inflammation of the iris. Several times the author has observed this complication. The exudation disappeared and the result was good, although at first the appearances were most alarming.

Bulging or Cystoid Cicatrix.—Instead of perfectly smooth healing, the cicatrix at the end of a week or two may begin to bulge, sometimes at one or other extremity of the wound, and sometimes through its entire length. The bulging consists in a vesicle-like, semitransparent elevation, and is generally associated with an entanglement of the iris in its margins, together with distortion of the coloboma. Eyes in which such entanglement of the iris has taken place are likely to develop iridokeratitis, and it has been recommended that the cystoid cicatrix should be removed and the opening closed by the application of the electro- or thermocautery. This procedure, however, has proved to be a dangerous one in several instances, and has been followed by severe inflammation and even by sympathetic ophthalmia.

Glaucoma after Extraction.—This complication occurs after a severe iritis, with numerous posterior synechiæ, which has led to the formation of a membrane. It may be the sequel of an iritis, which is characterized by a deep anterior chamber and dotted opacities on the cornea, or also of an iritis which is only slight in character, but where there has been an adherence of the pillars of the coloboma to the cicatrix and also to the lens-capsule, or where tags of capsule have attached themselves to the under surface of the corneal wound (see also page 392). This tends to obliterate the canal of Schlemm. Glaucoma may be caused by imperfections in technic and by obstruction caused by remnants of capsule and iris, and is prone to follow slow closure of the wound and the formation of an anterior synechia. Elschnig's investigations show that this condition may arise by reason of a proliferation of epithelial cells within the anterior chamber—that is, by an abnormal ingrowth from the anterior corneoscleral surface. Glaucoma of similar origin also occurs after the operation of laceration of the capsule—*i. e.*, after discission. If uncontrolled by miotics, iridectomy, sclerotomy, or cyclodialysis should be performed.

Opacities of the Cornea and Keratitis.—Opacity in the cornea may be due to the introduction of antiseptic fluid, especially solutions of bichlorid of mercury, into the anterior chamber. It has a peculiar, milky-white appearance, and is located chiefly at the posterior surface of the cornea, although the epithelium may also be rough. It does not

disappear, and, if sufficiently thick, entirely vitiates the effect of the operation.

This opacity must not be confounded with a very common type of keratitis occurring after cataract extractions, which has received the name *striated keratitis*, consisting of fine stripes of opacity radiating in several directions across the cornea. This entirely disappears in a few days, and need not give rise to apprehension. As Frederick Tooke points out in his admirable studies of the pathology of the corneal section, this condition is more common in elderly subjects than in those of younger years, and may be due to alterations in the endothelium or in Descemet's membrane dependent on age, which permit access of aqueous through the cells, the tension having been released by the corneal section. Occasionally at the end of a week or more *herpes of the cornea*, heralded by sharp pain and lacrimation, may develop, and from the herpetic spots small filaments may arise—*filamentous keratitis*. The lesions will subside under the influence of light bandages and antiseptic lotions; holocain is of much service.

Prolapse of the Iris.—This complication is the chief objection to the operation of simple extraction, and varies in frequency from 3 to 10 per cent., according to different statistics. The prolapse is usually heralded by a sudden sharp pain, which gradually passes away. It generally results from trauma—for example, striking the hand against the eye—or is due to a fit of coughing, violent exertion, straining effort, or similar cause. If the prolapse is discovered soon after its occurrence, —that is, at the first dressing—it should be cut off and the edges of the iris reduced, exactly as after the operation of iridectomy. If the prolapse is not noted until the third or fourth day, it is sometimes proper to allow it to remain. The eye should be firmly but gently bandaged and atropin may be instilled, although some surgeons prefer eserin. Small prolapses may disappear, others produce no irritation, while still others become larger, constricted at their bases or cystoid. H. Knapp allowed these to remain until the irritation had disappeared, and then amputated them in the same manner as a small staphyloma is abscised, and usually obtained smooth and permanent recovery. Occasionally iridocyclitis occurs, and sympathetic ophthalmitis has been reported. In general terms the safest procedure is to excise the prolapsed iris as soon after its discovery as is possible.

Prolapse of the iris after combined extraction—*i. e.*, entanglement of the edge of the cut iris in the angle of the wound—is not uncommon.

Delayed Restoration of the Anterior Chamber and Delayed Healing.—Often the wound after cataract extraction is closed at the end of twenty-four hours, usually not later than the third day. Occasionally, however, there is delayed restoration of the anterior chamber, which in most instances is caused by some foreign substance—for example, a particle of capsule or conjunctiva between the lips of the wound, or to an almost imperceptible incurvation of the flap (Trousseau). Doubtless, in most cases, an error in the technic of making the section is

responsible for slow closure of the wound. In a few instances the failure to unite appears to be due to excessive secretion of aqueous humor or to lack of reparative power, depending upon some anomaly in the condition of the patient. Derrick Vail attributes this complication to an involuntary spastic contraction of the orbicularis and has remedied it by severing the muscle, cutting directly upward and downward, from its external attachment. A conservative treatment is generally indicated, and it is usually recommended that bandaging and rest in bed shall be continued until the chamber is restored, but the author agrees with Berry that if any dressing be applied at all, if the wound does not close readily, it should be of the lightest character and should exert no pressure on the lids. Usually removal of the bandage and the adjustment of protecting spectacles (wire-gauze) facilitates the healing (Gifford). If a piece of capsule or other foreign substance can be detected, it should, of course, be removed. A light cauterization of the line of incision with a point of nitrate of silver or a probe dipped in carbolic acid is often of service. *Delayed union* of the wound, especially after corneal incisions, with separation of its margins, may end in recovery, but has been followed by infection and by glaucoma, especially if the lens capsule becomes adherent to the corneal wound. Associated with tardy or imperfect wound closure there may be a glossy edema of the conjunctiva in its lower part, which H. Knapp called *filtration chemosis*. It will subside when the union of the incision is firm.

Postoperative Delirium and Insanity.—Delirium after operation has been referred to. Sometimes marked dementia follows cataract extraction. The delirium has been ascribed to the use of the bandage, to the effect of atropin, to imperfect mental balance existing prior to the operation, and to auto-intoxication. If possible, the bandage should be removed and the patient given various sedatives, *e. g.*—the bromids—according to the indications. Paraldehyd is useful; morphin usually does not act well in these circumstances.

Choice of an Operation.—Obviously, the advantages of simple extraction are the absence of mutilation of the iris, and consequently the formation of a round pupil which reacts freely to the changes of light and shade and prevents the dazzling caused by the presence of a coloboma. Its disadvantages are the difficulty of expelling the lens, the increased difficulty of performing perfect toilet of the wound, and the danger of prolapse of the iris. In the judgment of the author certain cases require iridectomy—namely, those in which the ball is hard, the lens is large, the anterior chamber is shallow, the iris is not readily dilatable, or there is ciliary irritation. The combined method is also preferred if the cataract is not ripe or if the patient's mental or physical condition tends to create restlessness. In other circumstances simple extraction may be performed, and this was the author's practice until within the last few years. He has, however, returned to combined extraction with a small iridectomy, as it is, on the whole, a more satisfactory procedure. For the reasons already given the author is unable

to agree with those surgeons who believe extraction of the lens in its capsule should be the operation of choice.

Cataract extraction without iridectomy may be performed according to *Chandler's method*, in which a small piece of iris, 1 mm. in diameter, is removed, making a very small, round opening as near the root of the iris as possible. This facilitates drainage and prevents iris prolapse. This operation is recommended by C. Hess and also receives the commendation of Fuchs. In Angelucci's modification of cataract extraction fixation is on the superior rectus muscle, and the entire operation is completed without speculum or aid of assistant.

Preliminary Iridectomy.—Some operators, almost as a rule, perform a preliminary iridectomy and extract the cataract several weeks later, because by this method the dangers of the final operation are lessened. It is to be recommended in any case where serious complications are apprehended, where for any reason an extraction in one eye has terminated unfavorably, or where the cataract is not ripe.

Preliminary Capsulotomy.—Operative procedures designed for the purpose of ripening immature cataract are described on page 409. Homer E. Smith advises preliminary capsulotomy and operates as follows:

> The capsulotomy knife is thrust through the middle of the superotemporal quadrant of the right cornea and is made to reach the lowest point of the dilated pupil and to rest on the vertical meridian of the lens. The handle is now raised until the blade penetrates the capsule. Next, the handle is made to describe an arc of a circle away from the operator, the capsule being incised along the vertical meridian, while the shank is gradually withdrawn. The point of the knife is next released, the shank introduced, the blade carried to the nasal margin of the pupil, and the capsule incised along the horizontal meridian. If the left eye is to be operated upon, the knife is entered in the temporo-inferior quadrant of the cornea and the first incision is from above downward. Eserin (gr. $1\frac{1}{2}$ to f$\overline{3}$j—0.097 gm. to 30 c.c.) is instilled and the eye bandaged. At the expiration of six hours the lens is extracted in the usual manner. The object of this operation is to separate the capsule from the lens and bring about a union of the nucleus and of the cortex.

Smith's procedure has received the endorsement of a number of surgeons. Apparently there is no harm in extending the interval between the two operations to twenty-four hours (Hansell and Shannon).

Fig. 353.—Knapp's knife-needle.

Operations for After-cataract.—After-cataract—or, as it is usually called, *secondary cataract*—has been described. If it is a delicate, web-like membrane which stretches across the pupil, and which is best seen by artificial illumination—*i. e.*, by condensing with a large magnifying-glass a beam of light into the pupillary space—the treatment may consist in the introduction of a cataract needle in the manner described under Discission, and making a laceration in the membrane. The

author is accustomed to operate with Knapp's knife-needle (Fig. 353) in the manner advised by this surgeon—namely:

The pupil being dilated *ad maximum*, and the area of the operation being perfectly illuminated, the knife-needle is thrust through the cornea 3 mm. from its margin in the horizontal meridian. Next, the knife-needle is advanced to a point close to the opposite margin of the iris, where the membrane is punctured, and a horizontal incision of 4 or 5 mm. is made. This being accomplished, the point of the needle is raised toward the cornea and passed upward in front of the membrane, which it transfixes at a point 2 mm. above the horizontal incision, and divides it by a cutting movement downward, as far as the horizontal incision. The same procedure is performed on the lower half of the membrane, cutting from below upward. Thus, a crucial incision is formed, and if successful, the retraction of the edges leaves a good central aperture in the membrane. It is essential to cut the after-cataract, and not to tear it nor drag upon the ciliary body. Therefore the instrument should avoid thickened portions of the capsule. It is also desirable that it should not enter deeply into the vitreous.

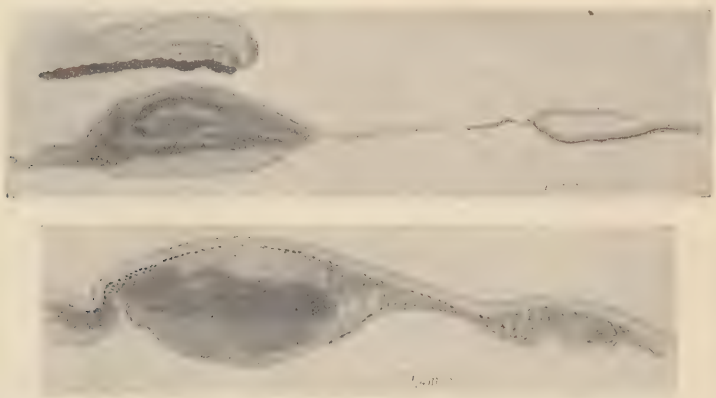


Fig. 354.—Sections of secondary cataract, showing inclusion of cortical remnants between the posterior and anterior capsule and curling of the capsule upon itself. (From a specimen prepared by Dr. C. M. Hosmer in the author's laboratory.)

In place of entering the knife through the cornea in the manner already described, the puncture may be made through the conjunctiva at the corneoscleral border, after the manner recommended by Kuhnt, a method which the author is accustomed to follow. In place of this operation, Ziegler's method (see page 627) may be employed; indeed, in recent years the author has used this admirable operation almost exclusively in all types of after-cataract.

Where the membrane is thick and there has been much proliferation of the epithelium, discission with a knife-needle, owing to the dense and resisting character of the tissues and the danger of dragging upon the ciliary body and iris, is a dangerous operation. In these circumstances iridotomy or Ziegler's operation should be employed (see page 627).

Other plans are to divide the capsule with delicate cannula scissors, or to cut the desired opening with an instrument which works on the principle of a punch. In place of entering the knife through the cornea or corneoscleral border it may be passed through the sclera 6 mm. behind the corneal margin, and pushed forward so that its point passes

through the membrane into the anterior chamber. The membrane is cut by causing the knife to make a sweeping movement from before backward.

Discission is an operation invested with many dangers. In no circumstances should there be rough handling; the discission instruments must be very sharp, and the operator must avoid dragging upon resisting bands. Preceding the operation and following it there should be the free use of atropin. If signs of reaction occur, the treatment of iritis is indicated.

Glaucoma after discission is an occasional complication, and is characterized by pain, steamy cornea, impaired vision, and increased tension. It should be treated by eserin locally, morphin and chloral internally, and, if these measures fail, by iridectomy or paracentesis and evacuation of the vitreous from the anterior chamber.

In cases of occlusion of the pupil by a drawing up of the iris, or where there are bands of strong inflammatory lymph, to which also the name secondary cataract is sometimes applied, discission is not advisable. In most instances iridotomy or V-shaped iridotomy with a knife-needle is the best operation.

OPERATIONS UPON THE EYE-MUSCLES

These consist of *complete* and *partial tenotomy* and *advancement* or *readjustment, advancement with muscle resection.* For the operation of tenotomy the following instruments are required: A stop speculum or lid-elevator, two strabismus hooks (Figs. 355, 356), fixation forceps, and a pair of probe-pointed scissors, the form devised by Dr. Jackson being particularly suitable. In young children general anesthesia may be necessary; but, if possible, cocain should be used. Usually the internal rectus is divided; quite frequently the external rectus; less commonly the other straight muscles.

Figs. 355, 356.—Strabismus hooks.

Complete Tenotomy.—In a tenotomy on the internal rectus, for example, the operator proceeds as follows:

The eyelids being separated with a stop speculum, the surgeon catches with a fine-toothed forceps a fold of conjunctiva and subjacent fascia on a level with the lower border of the tendon, and with the probe-pointed scissors makes an opening just large enough to admit the strabismus hook. He may with one clip divide conjunctiva, subjacent fascia, and the capsule of Tenon; otherwise, after the division of the conjunctiva and subconjunctival tissue, Tenon's capsule must be picked up and incised in a length equal to the cut made in the overlying structures. A strabismus hook is next passed behind the tendon, its point turned upward, and made to appear at the upper border of the tendon beneath the conjunctiva. It is next drawn forward and outward toward the cornea, and scissors, with their blades slightly parted, are introduced between the hook and the eye, and the tendon cut close to its sclerotic attachment. This is the *subconjunctival operation,* and was introduced by Critchett.

Instead, the subconjunctival method, especially in cases where there is a considerable squint, the open operation, or, as it is known, the Graefe method, may be performed as follows:

The operator seizes with fixation forceps a fold of conjunctiva and subconjunctival tissue parallel with the corneal margin over the insertion of the tendon and divides the tissue raised by the forceps horizontally down to the sclera. Next the point of a strabismus hook is pressed firmly against the sclera below and behind the insertion of the tendon, under which it is passed until it reaches its upper margin. With the hook in position the exposed tendon is put slightly upon the stretch and separated from its attachment by means of blunt-pointed scissors. The hook is next passed with its point turned above and below and any tendinous fibers which may have escaped are divided. The hook should now pass readily to the corneal margin. Sutures used for closing the wound should be inserted vertically, unless it is desired to lessen the effect of the operation, in which case they are placed in a horizontal direction.

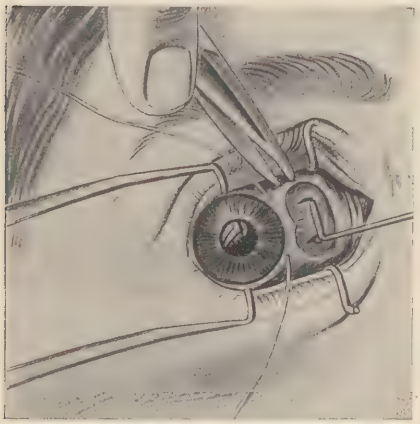


Fig. 357.—Exposure of the internal rectus tendon, which is lifted upon a hook (after Haab). This drawing also illustrates Prince's method of advancement (see page 677). The suture in the sclera, to which the tendon is afterward fastened, is seen lying along the corneal margin.

After tenotomy the conjunctival sac should be thoroughly irrigated with boric acid solution or bichlorid of mercury (1 : 10,000) and *both* eyes bandaged for a day or two. The conjunctival suture may then be removed and the patient wear his correcting glasses. If the patient is in suitable surroundings, a bandage may be dispensed with and the spectacles which correct the refractive error may be worn immediately after the operation. The latter procedure is followed by the best results.

Snellen's method of operating is satisfactory, and one which the author often employs. A small opening, about 4 mm. in width, is made through the conjunctiva over the insertion of the tendon, the center of which is then incised vertically. Through this opening the point of a strabismus hook is inserted and the upper and lower half of the tendon divided. A suture closes the conjunctival wound. Stevens' method, described on page 674, is a modification of this operation and may be used for complete as well as for partial tenotomies.

Tenotomy of the other straight muscles may be performed according to the methods already described, the operator remembering the distance of the insertion of each tendon from the corneal margin (see page 519).

Tenotomy of the Inferior Oblique.—This operation was originally suggested by E. Landolt, but its indications and technic have been especially elaborated by Duane. Posey, who has made a valuable contribution to this subject, thus describes the operation:

After injecting the tissues with a 2 per cent. solution of the novocain, a curvilinear incision is made just superior and parallel to the lower and inner bony rim

of the orbit, the tissues being divided down to the bone. A strabismus hook is next inserted, and gentle traction is made on the tissues until the muscle is encountered. This is assured by the upward movement imparted to the globe by traction on the hook. The tendon is next drawn forward and divided. The wound having been closed with one or two stitches, a firm dressing is applied and allowed to remain in place for forty-eight hours. Occasionally a subconjunctival ecchymosis appears at the lower and inner part of the globe.

The *indications* for this operation, based upon Duane's studies, are thus summarized by Posey:

(*a*) Complete stationary paralysis (especially congenital or traumatic) of the opposite eye.

(*b*) Partial paralysis of the superior rectus of the opposite eye if, owing to fixation being performed by the latter, the fellow eye develops a spasm of the inferior oblique, giving rise to diplopia, a disfiguring upshoot of the affected eye, or a lasting torticollis.

(*c*) Spasm of the inferior oblique, either secondary to paralysis of the superior oblique or some other muscle in the same eye, or occurring as a primary condition, provided the symptoms are sufficiently disturbing to warrant operation.

Complications in Tenotomy Operations.—1. The operator may fail to have divided the capsule of Tenon. In these circumstances he will also fail to introduce the hook beneath the tendon, and by such failure will recognize that he has not sufficiently incised the tissues.

2. *Hemorrhage.*—Occasionally severe hemorrhage follows a tenotomy, the blood rapidly pouring out beneath the capsule of Tenon and causing alarming proptosis. A firm pressure bandage should be applied, and gradually the proptosis will subside and the blood be absorbed.

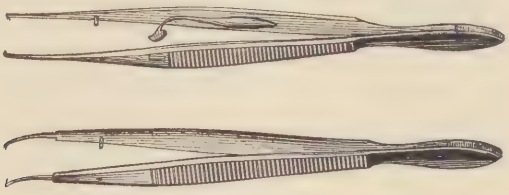
3. *Orbital Cellulitis and Tenonitis.*—Cellulitis has occurred from infection of the wound, the inflammation traveling back and causing an inflammation of the tissues of the orbit. The treatment of orbital cellulitis, described in another section, is indicated. Tenonitis, or inflammation of the orbito-ocular fascia, has followed squint operations.

4. *Perforation of the Sclera.*—Although this is a rare accident, it has happened to operators of considerable experience as the result of the use of sharp-pointed scissors, and for this reason the probe-pointed instrument is always to be preferred. In such circumstances the eye should be treated in the manner described on page 293.

5. *Retraction of the caruncle*, so that it sinks away from its normal position and gives a most disagreeable and peculiar stare to the eye, is a very unfortunate occurrence after a squint operation. A very slight degree of this is liable to occur even after the most careful tenotomy of the internus. Where it exists in great degree, it is due in part to excessive dissection of the tissues, and in part to retraction of the muscle. There are several methods of overcoming this defect, the essential character of which is the loosening up of the contracted tissues and stitching the caruncle into place.

Partial or Graduated Tenotomy.—Graduated tenotomies are performed for the purpose of correcting those conditions which are described under Heterophoria. The operation has been especially elaborated by Dr. Stevens, of New York, and is performed as follows:

With a pair of small, narrow-bladed scissors a transverse incision is made through the conjunctiva exactly corresponding to the line of insertion of the tendon. This is seized behind, but near its insertion, and a small opening is made dividing the center of the tendinous expansion exactly on the sclera. This opening is then enlarged by careful cuts with the scissors toward each edge, keeping carefully on the sclera as the border of the tendon is approached; the amount to be cut depends upon the



Figs. 358, 359.

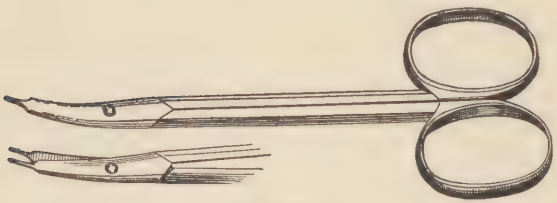


Fig. 360.

Figs. 361–363.

Figs. 358–363.—Stevens' instruments for tenotomy.

judgment of the operator and the need of the case, and is further regulated by placing the patient before a lighted candle and testing the sufficiency of the muscle upon which the operation is made, in the manner already described in connection with the investigation of heterophoria. In dealing with strabismus, the surgeon may determine to continue his section through the border, leaving uninjured, as far as possible, both the anterior and posterior lamellæ of the capsule, as well as the expansion at each border, to hold the muscle in relation to the eye. Turning the scissors then in the direction of the other border, this portion is dissected with equal care.

Figures 358 to 363 illustrate the delicate instruments which are used in this operation. They may with equal propriety be employed in ordinary tenotomies, and are satisfactory for this purpose, inas-

much as the laceration of the tissues is less marked, while the effect is equally great if the incisions are carried sufficiently far according to the directions already given.

Several other operative procedures for the purpose of elongating the tendon by partial tenotomies have been designed, especially by Ziegler, Verhoeff, and Todd. The first-named surgeon describes the characteristics of his operation as "complete division of each lateral third of the tendon and careful snipping of the superficial fibers in the central third, until sufficient elongation is obtained to yield a measurement of orthophoria."

Advancement or **readjustment** is an operation in which the tendon of a rectus muscle is brought forward to a new attachment. The operation is applicable to cases in which the tendon has become weakened, as, for instance, in myopia, together with the production of divergent squint; to those cases of convergent strabismus in which it is desirable to combine advancement of the external rectus with tenotomy of the internus; to free bilateral advancement to the exclusion of tenotomy; to certain cases of heterophoria (see page 561); and to cases in which an injudicious division of the internal rectus, for instance, has converted a convergent into a divergent squint. For other indications, see pages 551 and 552. General anesthesia may be necessary in young subjects and nervous patients.

The same instruments which are used in tenotomy are required, in addition to which suitable curved needles, a needle-holder, silk thread, fine catgut, and advancement forceps should be provided. Numerous methods of advancement have been designed. With the operation, a description of which follows, recommended by Swanzy, the author has achieved satisfactory results:

An opening is made in the conjunctiva immediately over the insertion of the tendon which is to be advanced, twice the breadth of the tendon. A band of conjunctiva between the opening and the cornea is next separated with the scissors from the sclerotic. A strabismus hook is now passed under the tendon, which is freely separated from the sclera; the hook is brought well up to the insertion of the tendon, care being taken that the whole width of the tendon is held on the hook. A curved needle carrying a strong black silk suture is introduced from its upper margin between the tendon and sclerotic, and passed through the tendon at its middle line. In the same way another suture is passed behind the tendon from its lower margin, and through it close to the first suture. Each of these sutures is knotted firmly on the tendon, a long end being left to each. For the strabismus hook Prince's advancement forceps is now substituted, which firmly grasps the tendon, which is next separated with scissors from the sclerotic close to its insertion. The needle on the end of each suture is next passed through the superficial layers of the sclera and beneath the conjunctival flap to the margin of the cornea in the manner illustrated in Fig. 365, and while an assistant rotates the eyeball toward the muscle which is to be advanced, each suture is tied with its own end. If there is redundant tissue, it is trimmed away and the conjunctiva sewed with three interrupted sutures over the advanced tendon, the central suture being passed through the conjunctiva and the advanced tendon to the margin of the cornea. Naturally, a greater or less effect is produced according as the sutures are placed farther from or nearer to the insertion of the tendon, and according to the extent to which the loosened tendon is drawn toward the corneal margin. Both eyes are bandaged and should remain

covered for at least four days, when the superficial sutures are removed. The deep sutures are allowed to remain, if they produce no irritation, from eight to ten days. An objection to this operation is the knot in the tendon, which must be removed through a small opening in the conjunctival surface, but if black silk is used the author has not experienced any difficulty; and it has seemed to him that the results were better than if the suture was not fastened in the manner described.

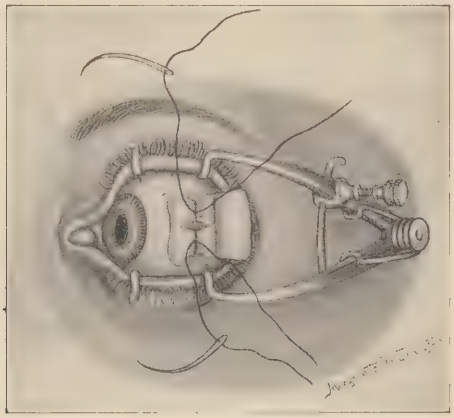


Fig. 364.—Advancement of the external rectus. The tendon has been exposed and the sutures tied upon it.

Landolt's Method of Advancement.—"The speculum having been adjusted, a conjunctival flap the summit of which reaches the edge of the cornea is cut and folded back so as to expose the insertion of the muscle which is to be advanced. Next a flattened hook is passed beneath the tendon and a second one in the opposite direction. The first hook is then withdrawn and the second intrusted to an assistant.

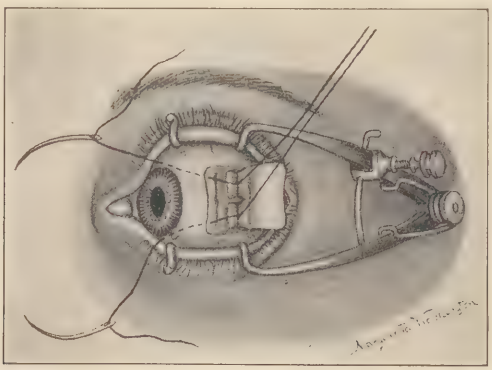


Fig. 365.—Advancement of the external rectus. The tendon has been separated from its scleral attachment, and the sutures will be passed through the scleral tissue beneath the conjunctiva in the direction of the broken line.

Two sutures are now introduced from without inward, about one-third of the width of the muscle from either edge. These sutures also include the surrounding tissues. In *simple advancement* the sutures are introduced immediately behind the hook, and the insertion of the muscle is detached from the ocular globe. In a *resection* the sutures are introduced further back and the muscle divided between them and the hook. In order to accomplish this the muscle is gently raised—at one part by

means of the four ends of the stitches, which the surgeon holds in his left hand, and at the other by the hook which the assistant holds—and the tendinous end separated from the eyeball. One of the needles is next passed above, and the other below, the meridian, into the *episcleral tissue* close to the corneal margin (*a–b*, Fig. 366), to the extent of several millimeters. If the needle does not penetrate sufficiently deep, it should be guided farther underneath the conjunctiva, and if it is feared that it has not a thorough grasp, it may be passed once more through the conjunctiva. The assistant now seizes the ocular globe with a fixation forceps at the level of the antagonistic muscle, and rotates it toward the muscle which is to be advanced, while the surgeon ties the sutures, one of which is composed of white silk and the other of black silk. Both eyes are bandaged for five days in divergent, and for a week in convergent, strabismus. The sutures are usually removed on the sixth day."

In A. E. Prince's method of advancement an unyielding fixation point is obtained by utilizing the dense episcleral tissue, severing the muscle, and regulating the effect by a "pulley suture." In Schweig-

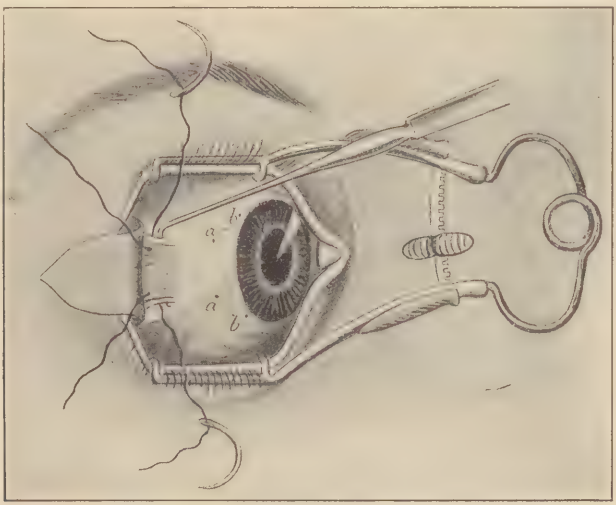


Fig. 366.—Landolt's method of advancement.

ger's method a free exposure of the muscle is made, and after the tendon is divided a portion of the end is resected; catgut sutures are employed to advance the muscle (see Reese's operation, page 678). H. D. Bruns, of New Orleans, has recently described an ingenious operation for advancement of the recti tendons, performed with the aid of a Clark hook and the formation of a tuck in the tendon, which is firmly flattened down and drawn strongly forward, and held in place by a combination of pulley and guy suture.[1]

Todd, after exposing a considerable portion of the tendon by means of a flap incision through the conjunctiva and capsule, forms, with the aid of an instrument known as the tendon folder, an actual folding, and fixes the duplicature with catgut sutures reinforced with silk sutures, which include the conjunctival flap. Major R. O'Connor has designed

[1] For the method of performing this operation, see Ophthalmic Record, June, 1903.

an ingenious and new operative procedure for shortening and lengthening ocular muscles.[1]

In Worth's method of advancement the needles carrying the sutures pass through the conjunctiva, capsule, and muscle. The main sutures ultimately are passed through the sclerotic, the needles traversing at least one-half the thickness of the sclerotic.[2]

Of the methods of advancement which have been recorded, the author is in the habit of employing the one which is described first and Landolt's procedure; he also finds Worth's operation exceedingly valuable.

Reese's Muscle-resection Operation.—Dr. Robert G. Reese operates as follows, the description of the operation being in his own words:

For the external and internal rectus make a vertical incision in the *conjunctiva* 6 mm. from the corneoscleral margin, commencing at the level of the upper corneal border, and extending to the horizontal plane of the lower border.

Fig. 367.—Reese's forceps for operation in squint.

At the upper and lower limits of the incision just made grasp the tissue anterior to the sclera with forceps and open with scissors, directing their point away from the muscle. This procedure allows the passage of the strabismus hook under the entire muscle.

When the muscle is held on the hook, dissect all the conjunctival and subconjunctival tissue back to the canthus, exposing the bare muscle completely.

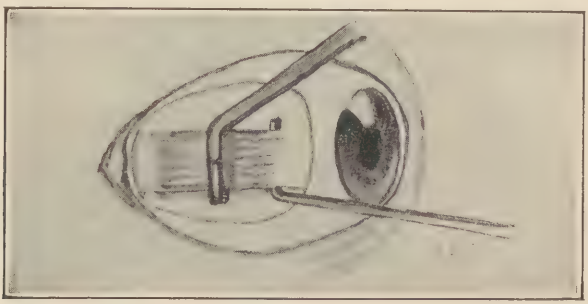


Fig. 368.—Passage of the strabismus hook under the entire muscle (Reese).

The lateral invaginations of Tenon's capsule, which are attached to the tendons of the ocular muscles, must be dissected free and clear.

One blade of the resection forceps is then inserted beneath the muscle at a right angle to its course, so that the groove on the blade lies directly over the middle

[1] For the method of operating, see Ophthalmic Record, December, 1914.

[2] For the method of operating, see Squint: its Causes, Pathology, and Treatment, 4th edition, by Claud Worth.

fibers of the muscle. Clamp the forceps to the last notch, and do not let its grasp include anything but muscle.

Sever the muscle 2 mm. from its scleral attachment, leaving a stump, so that the resected end can be sewed to its original insertion. Free the belly of the muscle from any scleral adhesions. Three sutures are necessary.

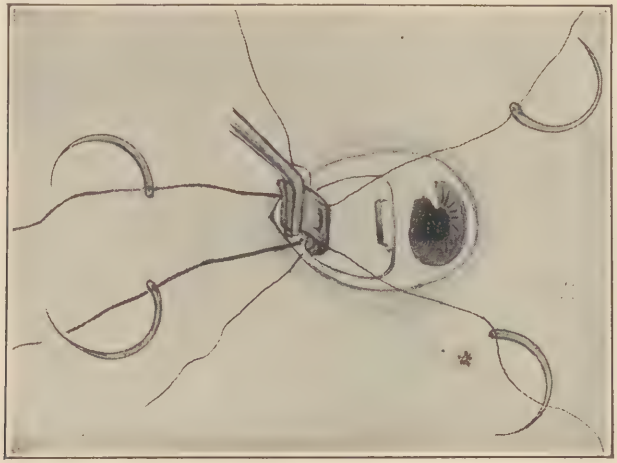


Fig. 369.—Showing the three necessary sutures (Reese).

Put the sutures in, commencing with the middle, which is a No. 3 braided silk with a needle on each end. Pass one needle through the scleral surface of the muscle posterior to the blade of the forceps and 4 mm. back of the point of resection, and

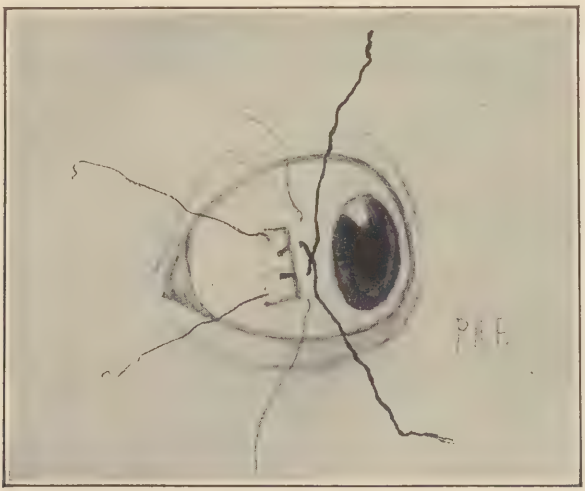


Fig. 370.—Showing the muscle resected and sutures in scleral stump (Reese).

1 mm. to the side of the groove on the forceps; then pass the other needle the same way, but to the other side of the groove, making a loop with the suture on the scleral surface of the muscle. As the needles pierce the muscle, let them include the dissected edge of subconjunctival and conjunctival tissues.

The two wing sutures are No. 5 silk with a single needle passed *first* through the upper and lower part of the dissected conjunctiva and episcleral tissues, including the superior and inferior border of the muscle, and slightly posterior to the loop made by the middle suture.

Cut the muscle anterior to the sutures, leaving at least 2 mm. in front of the loop.

Insert the two needles attached to the middle suture 2 mm. apart, through the center, and the other two needles through the upper and lower edges of the scleral stump. These needles should include the conjunctiva as they pass from behind forward.

Tie the middle suture first in a loop, and do not use a surgeon's knot, as it will not pull up well. The lateral sutures are next tied. No supplementary conjunctival sutures are necessary.

The middle suture is removed in ten days, and the others can be taken out any time after forty-eight hours, or if left in they soon fall out.

The eye operated upon only is bandaged and is dressed daily for five days, when boric acid bathing, three times a day, is ordered; and if the eye is not overcorrected, the correcting lenses are ordered for constant use. On the other hand, if there is an undercorrection, a mydriatic is used and the proper glass is worn constantly.

The operation consists in resecting the muscle only, and not cutting out any of the other tissues of the eye. It is a myectomy, because in no degree of squint, however slight, will removing the tendinous portion of the muscle be sufficient.

Operation for Shortening the Tendon.—G. C. Savage and Francis Valk secure the advantages of advancement by an operation in which the tendon is shortened. The last-named surgeon operates as follows:

"The conjunctiva is raised with forceps over the lower or upper point of the insertion of the tendon, and a vertical incision followed by a horizontal one, forming an L, is made. This is dissected loose from the underlying tissue, and then an opening is made in Tenon's capsule and a small hook is passed beneath the tendon. As the point of the hook comes out, another hook is inserted in an opposite direction, and the two hooks forcibly drawn apart, thus exposing the tendon and part of the muscle. Next a small instrument called a twin strabismus hook is passed beneath the muscle, and the hooks are allowed to separate by the action of a small spring in the joint, and the two hooks are then removed. The muscle and the tendon are now fully exposed and ready for the future. A needle threaded with catgut is passed first through the lower part of the tendon, then through the muscle as far backward as it is desired to make the 'tuck,' passing from within outward. It then goes across the belly of the muscle and is passed through, from without inward and back to the tendon, where it passes from within outward, at a point corresponding to its first insertion. As the ends are tied over the tendon at this point it is easy to see the 'tuck' formed as the muscle-belly is drawn forward and its long axis shortened."

Advancement of the capsule of Tenon is recommended by some surgeons. S. Lewis Ziegler has designed a *capsulomuscular advancement with partial resection*. His method is as follows:

A vertical incision is made in the conjunctiva near the limbus and the tendon and muscle raised on two tenotomy hooks introduced from below upward and put slightly on a stretch. One arm of a double armed suture is then inserted through the lower edge of the muscle from before backward and the same suture repeated behind the first, thus making a marginal whip-stitch. The thread is then passed partly across the top of the muscle and a similar double stitch is duplicated on the upper margin of the muscle.

Each needle is now carried backward in a parallel line beneath Tenon's capsule and made to emerge on the conjunctival surface. A small wedge or V-shaped piece

is then cut away from each peripheral third of the muscle, above and below, with punch or scissors, leaving a central strand of fibers intact.

The needles are now entered through the conjunctiva at the upper and lower extremities of the wound, passed firmly into the sclera for solid anchorage, and brought out near the limbus, where the suture is tied after the toilet of the wound has been performed. This leaves a knot and two parallel lines of thread exposed on the conjunctival surface, thus holding all the tissue flat against the sclera. The good judgment of the operator must decide how much capsule shall be engaged and just how tight the graduated suture shall be drawn. As a rule, the squint should be slightly overcorrected. If indicated, certain measures should also be taken to weaken the pull of the antagonist.

OPERATIONS UPON THE LACRIMAL APPARATUS

Slitting the Canaliculus.—This is performed as follows:

The lid being drawn down and out with the thumb, and the canaliculus knife held vertically, the probe point is introduced into the punctum. The handle is now

Fig. 371.—Weber's canaliculus knife.

depressed into the horizontal position, and the instrument pushed along the canal until the probe point touches the inner wall of the lacrimal sac. It is then raised to the vertical line with the cutting blade turned slightly inward, and the roof of the canaliculus divided. Either the upper or the lower canaliculus may be slit.

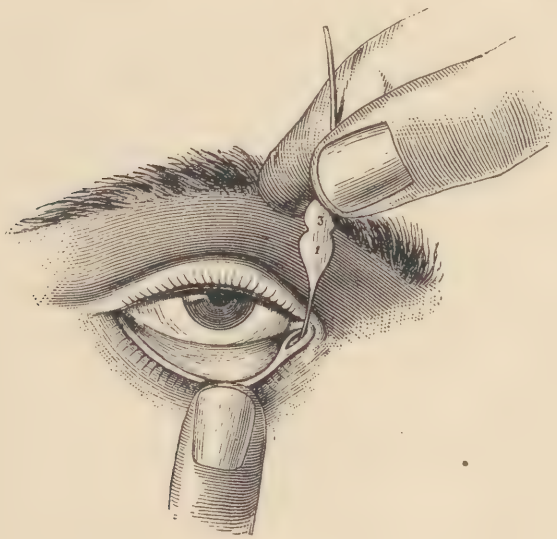


Fig. 372.—Introduction of a lacrimal probe (Meyer).

Introduction of the Lacrimal Probe.—The probe (Bowman's or Williams' probes are commonly employed; modifications have been devised by Theobald and Tansley) is introduced by passing it horizontally

along the canaliculus until its point touches the lacrimal bone. It is raised to the vertical position and pushed into the duct, remembering that the direction should be downward, slightly backward, and outward (Fig. 372).

Incision of a Stricture.—If the stricture resists, it may be divided with a knife, either the one which has been employed in slitting the canaliculus or, still better, with the instrument of Stilling. The knife is introduced in the same way as the probe, pushed down into the duct, and the stricture incised. The knife is next partially withdrawn, turned slightly, and the maneuver repeated. Dr. Charles Hermon Thomas,

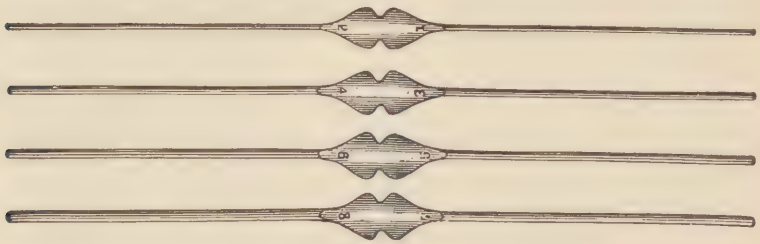


Fig. 373.—Lacrimal probes.

of Philadelphia, has devised a special knife, or *stricturotome*, which may be utilized for this purpose.

Introduction of the Lacrimal Syringe.—The nozzle of an Anel syringe can be introduced along the canaliculus without slitting it. The lid is drawn down and outward in the same manner as if the operation of slitting the canaliculus were to be performed, and the point of the syringe introduced. Sometimes the punctum is swollen shut and the nozzle cannot be inserted. In these circumstances the punctum may be dilated with a silver pin. Ordinarily a lacrimal syringe is furnished with a cannula probe. This is introduced into the duct in pre-

Fig. 374.—Thomas' stricturotome.

cisely the same manner as the solid probe; the syringe is filled with an antiseptic fluid, inserted into the mouth of the cannula, and the liquid injected into the duct.

Excision of the Lacrimal Sac.—In order to meet the indications described on page 572, excision of the lacrimal sac may be performed as follows:

After thorough cleansing of the sac through the canaliculus with a 1 : 10,000 bichlorid of mercury solution, general anesthesia may be induced, although in most instances careful local anesthesia will be sufficient (see page 595). With the skin drawn toward the bridge of the nose, the surgeon makes a slightly curved incision down to the periosteum, which extends from 4 mm. above the internal palpebral

ligament to 5 mm. below it, its length being 2½ cm. The canthal ligament may or may not be divided with scissors, and while the lips of the wound are separated, the temporal lip being especially drawn outward, the fibrous expansion from the tendo oculi is divided through its whole length, exposing the sac, which usually can be recognized by its bluish color. The sac is next gradually separated from the periosteum, being dissected out very much in the manner of removing a cyst, care being

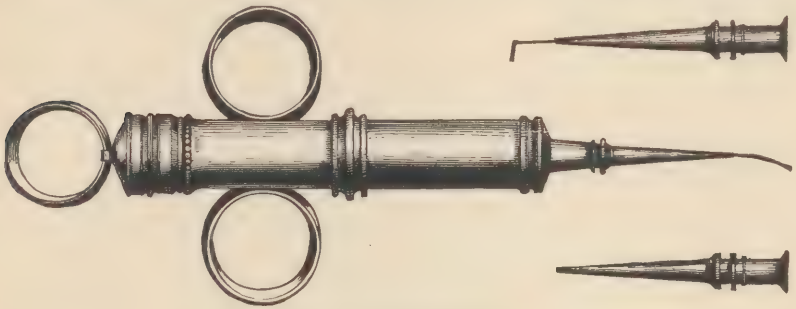


Fig. 375.—Anel syringe.

taken not to rupture its walls. The internal surface, the upper end and the posterior surface of the sac having been freed, is cut through at the commencement of the nasal duct. Sometimes the field of observation is obscured by a smart hemorrhage, which usually can be controlled by pressure or by specially devised specula; for example, those introduced by Axenfeld, or even more satisfactorily with the retractor designed by Bishop Harmon. Should the operator experience any difficulty in outlining the sac, its position may be localized by inserting a strabismus hook through

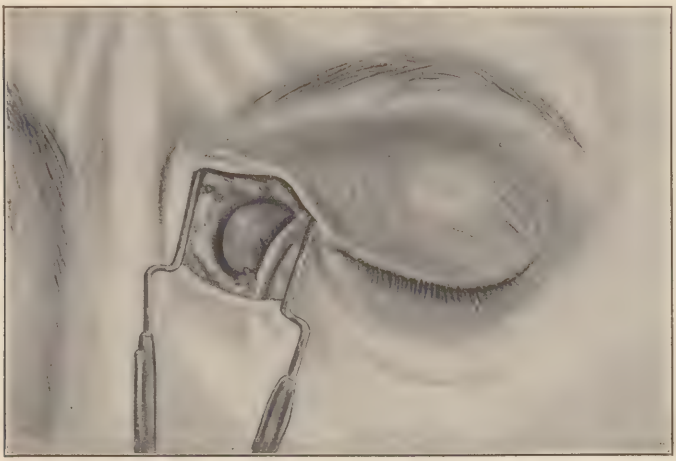


Fig. 376.—Extirpation of the lacrimal sac (Haab).

the canaliculus into the sac and keeping it there during the operation. This method was introduced by E. A. Shumway, and is most satisfactory in practice. Some surgeons advise that the sac shall be filled with melted paraffin prior to the operation, a procedure which the author has never found to be necessary.

C. R. Holmes does not believe that division of the tendo oculi is required in order to expose the sac, but dissects out the sac from underneath the tendon. If the tendo oculi has been severed, it may be replaced or repaired by a strong suture.

Great care must be taken that every portion of the sac is removed, and the operation may be terminated by thoroughly cureting the region (which Meller regards as unnecessary if the technic has been correct) and the ductus ad nasum, removing all traces of mucous membrane. Two sutures close the wound, which usually heals promptly. Holmes advises that the canaliculi should also be destroyed. Otherwise a blind pocket forms at the inner canthus. In order to accomplish this he splits the canaliculi through their entire length and destroys their lining membrane with the actual cautery. The dressing should consist of a pressure bandage placed over a light compress.

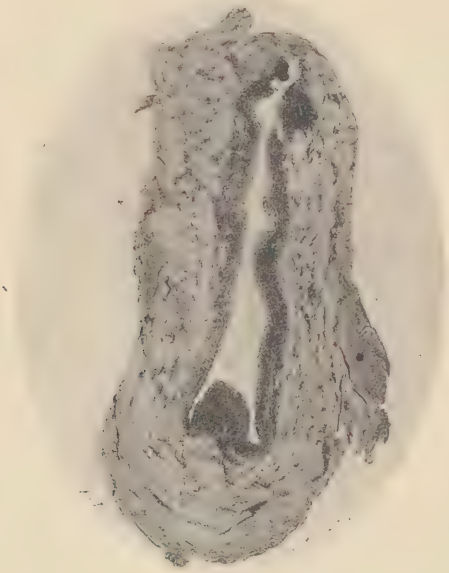


Fig. 377.—Excised lacrimal sac. Dense infiltration of mucosa with round cells; erosion and degeneration of the epithelium; sac wall densely fibrous and vessels engorged. Insane patient (Philadelphia General Hospital).

Extirpation of the Lacrimal Gland.—Following the direction of C. R. Holmes, this may be performed as follows:

An incision beginning near the center of the upper orbital arch and following the bony margin is carried to a point 3 mm. below the outer canthus. Next the fascia or septum orbitale is cut through along its attachment to the orbital margin. Should fatty tissue present in the wound, it must be held to one side with retractors and all bleeding from the edge of the wound must be controlled before the gland is separated from its surroundings, inasmuch as it is sometimes very difficult to distinguish the gland from the surrounding fatty tissue. By means of blunt-pointed scissors, fixation forceps, a small knife, and tenotomy hooks the dissection of the gland can be accomplished, and it may be removed without leaving any portion of it behind. Before the wound is closed all bleeding must be stopped. The lips of the wound are united with interrupted silk sutures, and the usual antiseptic dressing applied. As complications, hemorrhage into the orbit and atrophy of the optic nerve have been reported, and on a number of occasions a persisting conjunctivitis, and also, as, for example, in Veasey's case, a form of keratitis.

Extirpation of the Palpebral Portion of the Lacrimal Gland.—Instead of the removal of the orbital lacrimal gland, extirpation of the palpebral gland is often practised. It is a much simpler operation and may be performed as follows:

Thorough local anesthesia having been secured, the upper lid is everted and drawn upward from the eyeball while the patient looks strongly downward. This exposes the palpebral gland, which may be seized with toothed forceps and drawn outward. Its conjunctival covering is next incised, and the gland dissected from its surroundings. Hemorrhage having been controlled, the wound may be closed with one or two interrupted silk or catgut sutures, the upper lid replaced, and a light pressure bandage applied. The stitches are removed on the third day.

Toti's Operation (*Dacryocystorhinostomia*).—In this operation a passageway for the tears is made through the bony wall of the nose, and its main points are briefly summarized by Török, who recommends it as a valuable procedure, as follows: After chiselling through the bony lacrimal fossa, the internal wall of the lacrimal sac is resected, and at the same time a part of the mucous membrane of the nose is removed, which is of approximately the same size as the remaining external wall of the sac. The edges of the two mucous membranes are now brought into close connection, so as to permit their growing together. The mucous membrane of the lacrimal sac takes the place of the removed nasal mucous membrane. Together they now form one continuous mucous membrane, and with that the closing of the defect is prevented. The author has had no experience with this operation.

West's Operation (*Window Resection of the Nasal Duct in Stenosis*).—This consists in resecting, under local anesthesia, a window from the nasal duct in the upper part of the nose above the inferior turbinate, and involves the removal of part of the lacrimal bone and also a piece from the superior maxilla. This removes a stricture in the upper part of the duct, but leaves a stenosis in the lower part untouched. It is, according to J. M. West, who has designed this operation and whose description is quoted, immaterial whether the lower part of the duct is stenosed, so long as the tears can drain through the artificial window. Previous to operation a probe is passed into the duct to act as a guide. The author has had no experience with this operation.

APPENDIX

The Use of the Ophthalmometer.—Ophthalmometry or, more properly, keratometry has been briefly referred to on page 110. A number of new models of the Javal-Schiötz ophthalmometer are now obtainable, with variations in the disk, form of arm, and method of illumination, but they do not introduce radical changes. The following rules, prepared by the late Dr. E. W. Stevens, formerly associated with

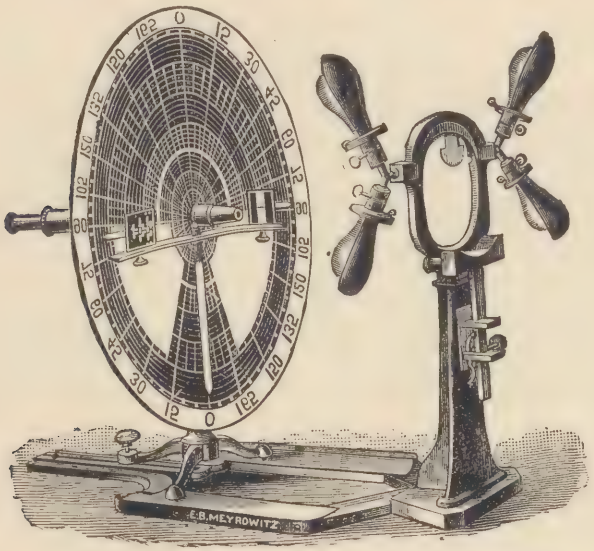


Fig. 378.—Javal-Schiötz ophthalmometer (old model).

the author in the Philadelphia Polyclinic, will enable the student to understand the proper method of using this instrument, as it is illustrated in Fig. 378:

The examiner, after satisfying himself that the illumination from the electric bulb is accurate, should carefully adjust the telescope by looking through it and turning the eye-piece either to the right or the left until the cross-hairs are brought clearly into view. The telescope is then turned so that the long pointer is below and at zero. The stationary mire on the parallelogram (Fig. 379, *A*) should be examined to see that it is in proper position, which is at 20° on the graduated arc.

The patient is now seated before the instrument in an easy position, with his chin resting on the chin-rest and his forehead pressed against the forehead-rest. His eyes should be widely opened and exactly horizontal—points to be determined by sighting through the transverse slit above the telescope. One eye is now covered with a small shade, and the observer sights along the telescope, through the notch

above it, at the patient's eyebrow; then, sighting through the tube, he moves the instrument forward or backward and raises or lowers it by the thumb-screw until the eye is brought into the field of the telescope, and a distinct image of the disk and mires is seen on the cornea.

The images of the disk are doubled, and, overlapping each other, form an oval space in which are seen the two mires or targets, to which the beginner should confine his attention. The observer now slides the mire at his right along the arc until its reflection touches the reflection of the stationary mire, and notes whether the two lines bisecting the two mires are continuous. If these two lines are not continuous, the telescope is turned so that the long pointer will move from 0° toward 135°. If the transverse lines do not become continuous when 135° is reached, the rotation proceeds no farther in this direction, but the long pointer is turned back to 0° and then toward 45°, but never beyond 45°. With regular astigmatism the lines always become continuous within 45° of 0°. When the lines are continuous the mires must be brought into perfect approximation (Fig. 379, 1). This is the *primary position,* which should be carefully recorded according to the position of the long pointer.

The telescope is next turned so that the long pointer moves 90° to the left of the primary position—*i. e.,* to a point which is known as the *second position.*

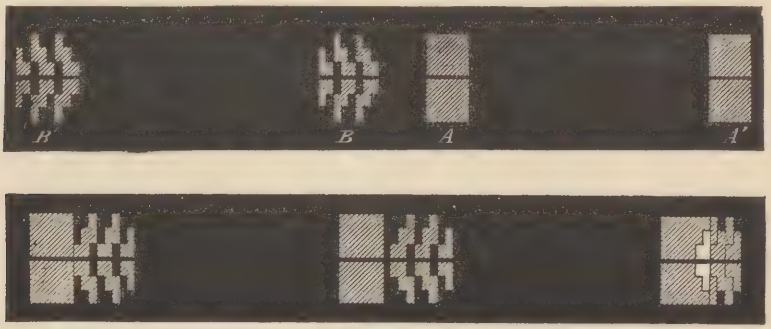


Fig. 379.—The mires.

If the mires overlap (Fig. 379, 3)—for example, two steps in the second position with the long pointer at 90°—there is astigmatism of 2.00 D with the rule, because each step is equivalent to 1 diopter of corneal refraction, and this is recorded + 2.00 D cyl., axis 90°, or − 2.00 D cyl., axis 180°.

If, on the other hand, the mires separate (Fig. 379, 2) in the second position, there is astigmatism against the rule. For example, if the primary position is found at 30°, and when the tube is turned to the left until the long pointer reaches 120° a separation of one step has occurred, there is astigmatism of 1 diopter against the rule, which is recorded + 1.00 D cyl., axis 30°, or − 1.00 D cyl., axis 120°.

In order to ascertain the exact number of steps to which the separation of the mires in the second position is equivalent, they are approximated by moving the sliding mire until the reflections touch, and the telescope is then rotated back to the primary position. The mires will now overlap, and the amount of astigmatism can be read off just as in astigmatism with the rule. The observer should remember, in finding the primary position, not to turn the long pointer farther than 45° on each side of 0° at the lower margin of the disk, lest he record astigmatism against the rule when it is with the rule, and vice versâ.

The upper surface of the arc carrying the mires is graduated on its outer circle to show diopters of refraction. It does not give the hyperopia or myopia of the eye, but indicates the corneal curvature. On the clamp of each mire there is a mark which enables one to read at a glance from this graduated arc the total refraction of each meridian of the cornea. The total refraction of at least one corneal meridian

should be recorded, and preferably the one of least refraction. For example, if the examiner finds in the right eye 1 diopter of astigmatism with the rule, the long pointer being at 75° in the second position, and the right-hand mire at 23° on the graduated arc, the refraction may be recorded O. D. 43.00 D = 1.00 D cyl., axis 75° with the rule.

If so desired, the astigmatism can be read from the graduated arc by measuring alternately the meridians of greatest and least refraction of the cornea.

On the right of the inner circle of the arc there is a scale graduated from 6 to 10, each space being divided into ten equal parts. These spaces record the radius of curvature of the cornea in millimeters, and the amount is indicated by a mark on the clamp of the traveling mire.

In some eyes it is impossible to bring into a continuous line the two lines bisecting the mires of the ophthalmometer, owing to irregular astigmatism or conical cornea. In these cases, however, the instrument is perhaps superior to all other methods of corneal measurement, as the overlapping or separation of the mires

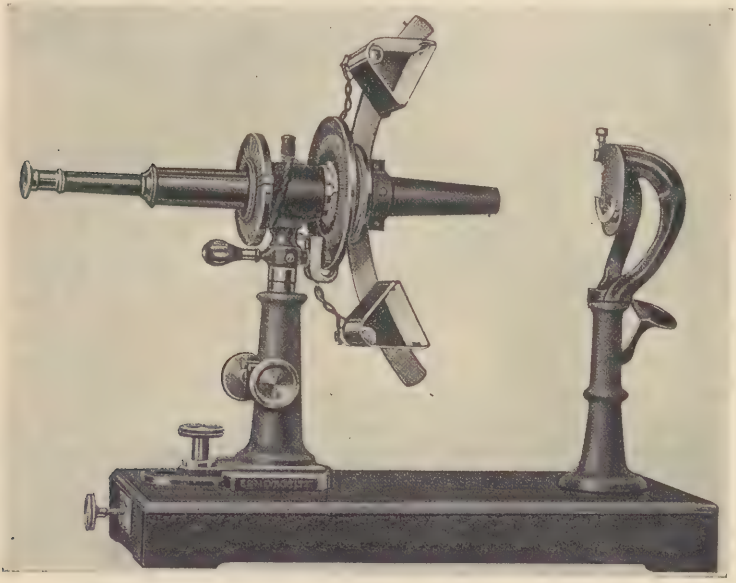


Fig. 380.—Javal-Schiötz ophthalmometer (model of 1907).

gives a clue to the axes of the meridians of least and greatest corneal curvature, as well as the amount of astigmatism.

Not infrequently the instrument indicates that the principal meridians of the cornea are not at right angles to each other—for example, it may record + 3.00 D cyl., axis 80°, or − 3.00 cyl., axis 180°. In these cases, when there is hyperopia, the axis of the cylinder should be 80°, and when there is myopia, 180°.

In patients with heavy overhanging lids, deep-set eyes, or long lashes it is at times extremely difficult or even impossible to measure the vertical meridian of the cornea with the ophthalmometer.

Nothing is more common than to see the mires separate and overlap again, so that the apparent curvature of the cornea seems to change while under observation. This change is due to slight movements of the eye which bring different portions of the cornea into view. It is difficult for most patients to remain long in the required position before the instrument, and hence the readings should be rapid as well as accurate.

As to the correspondence between the amount of corneal astigmatism indicated by the ophthalmometer and the total astigmatism under a mydriatic, there is a difference of opinion among observers. Probably the rule formulated by Burnett is, in the main, correct: "For the total subjective astigmatism, subtract 0.50 D from the corneal astigmatism when it is according to the rule, and add 0.50 D if the corneal astigmatism is against the rule."

In addition to the Javal ophthalmometer, a number of excellent models may be obtained. To some of these brief reference has been made on page 111. In so far as the author's practice is concerned, his best results have been obtained with the Javal instrument (Fig. 380).

The ophthalmometer is exceedingly useful, and one of the most important of all the instruments of precision we possess for the diagnosis of

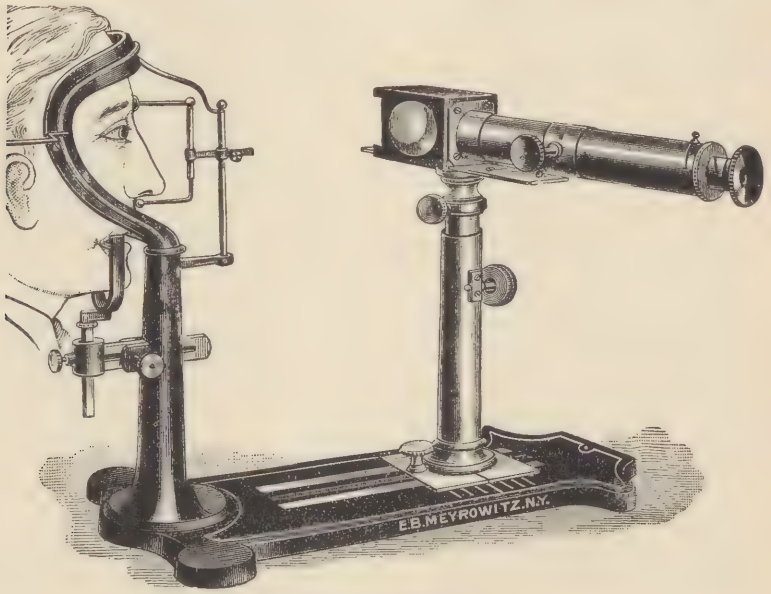


Fig. 381.—The tropometer.

astigmatism of the cornea; but it should never be used for the prescription of glasses to the exclusion of other methods—the trial-lenses after mydriasis, and retinoscopy.[1]

The Use of the Tropometer.—Dr. G. T. Stevens[2] attaches special importance to the determinations, absolute as well as comparative, of the rotations of the eyes, since he believes that excessive tensions upon the vertically acting muscles of the eyes often induce converging or diverging strabismus, independently of any anomalous tension of the laterally acting muscles, and that many conditions of heterophoria may be explained in a like manner.

[1] For a thorough exposition of the principles of keratometry the student should consult Carl Weiland, Archives of Ophthalmology, vol. xxii, pp. 37-64; Optique Physiologique, by Tscherning, pp. 46–68.

[2] International Ophthalmological Congress, Edinburgh, August, 1894; Annales d'Oculistique, April and June, 1895.

The most favorable rotations, according to Dr. Stevens, are: upward, 33°; downward, 50°; inward, 55°; outward, 50° (compare with page 522).

He has devised an instrument, called the tropometer (Fig. 381), for the determination of the various rotations, a description of which, kindly revised by Dr. Stevens, follows:

The instrument consists essentially of a telescope in which an inverted image of the eye is found at the eye-piece, where its movements can be observed upon a

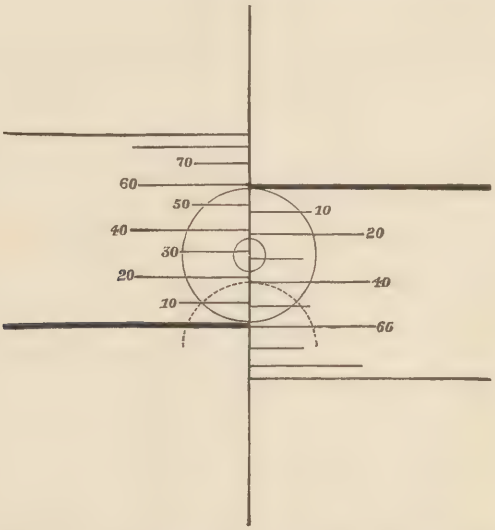


Fig. 382.—The long line between and at right angles to the shorter lines divides two similarly graduated scales running in different directions. The larger circle represents the outer border of the cornea, the edges of which are in contact with the two strong lines. The interval between each pair of short lines of the scale is ten degrees of an arc, commencing at the strong line in each case. If, now, the head of the person examined is held firmly in the primary position, and the eye caused to rotate strongly in a given direction, the arc through which the border of the cornea passes may be accurately read upon the scale. In the figure the curved dotted line represents a new position of the border of the cornea. Suppose that the person examined has been directed to look strongly upward. Then the cornea has moved *down* the scale, and reaches the point in this example of 40°, that being the measure of this rotation.

By means of the small lever the scale can be placed horizontally, vertically, or obliquely, and by means of the two graduations measurements in opposite directions can be made.

If it is desired to determine the upward rotation, the border of the cornea is made to coincide with the strong line which appears in the upper part of the scale at the right hand. This adjustment is made by means of the milled head at the side of the standard. As the eye rotates up, the image moves apparently down. In determining the downward rotation the strong line at the lower left-hand side of the scale is taken as the point of departure. For lateral rotations the scale is turned to the horizontal position, and the corresponding strong lines used as before.

graduated scale, permitting rotations in any direction to be measured. A prism or a diagonal mirror at the objective end of the telescope permits the observer to sit at the side of the observed. By means of a head-rest and an adjustable stirrup with a wooden bar, which the observed holds closely between the teeth, the head may be held firmly in the primary position. This position is indicated by the two buttons at the extremities of the guiding rods.

In order to adjust the upper border of the cornea to the line, it will generally be necessary for the examiner to place the left hand upon the forehead of the patient

and make gentle traction of the upper eyelid by the thumb. An application of the hand to the head is advisable in all measurements, as by this means the examiner is able to detect even a slight movement of the head, which would vitiate any measurement of the rotation.

In adjusting the head to the head-rest the teeth should be closed upon the wooden bar of the stirrup with force; then, after adjusting the stirrup to the proper height, the two indicators should be adjusted, one touching the glabella or ridge just above the root of the nose, the other pressing the commissure of the upper lip close below the nose. By pushing the stirrup forward or backward the lower indicatory button should be at a distance from the bone equal to that of the upper indicator.

The hoop passing around the head is designed to indicate, when the knob presses against the occipital protuberance, that the head is in position for lateral measurements.

If the cornea is large, the telescope must be moved backward upon the base until the borders of the cornea just encroach upon the two strong lines of the scale. When the cornea is small the tube is moved forward.

The wooden bar of the stirrup may be thrown away after use and replaced by another.

For measuring declinations of the retinal meridians, Dr. Stevens has designed an instrument known as the *clinoscope*. For a full description of this instrument and the manner of using it the student should consult the Medical Record, February 16, 1901, where he will find Dr. Stevens' complete directions.

Localization of Foreign Bodies in the Eyeball with the Röntgen Rays.—The following paragraphs have been written by Dr. William M. Sweet, and, therefore, in his own words describe the method which he has originated and which has proved to be most satisfactory:

The methods of locating foreign bodies in the eyeball by means of the Röntgen rays are based upon the study of the shadow of the foreign substance on the radiograph in its relation to the shadow of one or more known points in the vicinity of the eyeball. These fixed points from which measurements are made may be situated on the skin of the eyelid or cheek, or suspended in front of the eyeball. If an apparatus is employed to fix the position of the *x*-ray tube at each exposure, only one indicating point will be required, but with two fixed points of measurement the position of the tube at the time the radiographs are made need not be known.

The earlier form of the Sweet localizing apparatus consisted of a small platform to which the head of the patient was firmly clamped. Two ball-pointed indicator rods were used, one opposite the center of the cornea, and the other at a fixed distance to the temporal side. Two exposures were made, and the situation of the steel in the eye was determined from a study of the position of the shadow of the foreign body on the two plates in relation to the shadow of the two indicating rods.

A new form of apparatus has been designed, in which only one indicating rod is used. The planes of shadow of the foreign body are accurately determined by the instrument without the necessity on the part of the operator of taking measurements from the plates or in drawing lines on the chart. The tube-holder, indicating ball, and plate-holder are upon a movable stage, and therefore preserve a known relation to each

other which does not vary. The angle of the rays with the eyeball and the distance of the tube from the plate are always the same, so that one indicator is sufficient, and this consists of a small steel ball supported in a ring of transparent celluloid. The setting of this ball opposite the center of the cornea is made by means of adjusting screws conveniently placed on the frame of the instrument. Accuracy in the measurement of the distance of the indicating ball from the center of the cornea is secured by means of a telescope and reflecting mirror. The mirror gives an image of a cross-wire and a lateral image of the cornea. Through the telescope the observer adjusts the instrument until the image of the cross-wire is in direct contact with the image of the summit of the cornea (Fig. 383). When the adjustment is made, the indicating ball is exactly 10 mm. from the center of the cornea. A miniature incandescent lamp, mounted in an adjustable shade, illuminates the side of the nose of the patient, insuring a well-lighted image of the cornea and cross-wire.

Instead of a ball of cotton or other object for fixation, as in the older method, a circular mirror is placed at a distance of 12 inches above the injured eye. The patient gazes in the mirror and sees a reflected image of the injured eye and the circular celluloid disk with the steel indicating ball in its center. After the ball has been adjusted to a point opposite the center of the cornea of the injured eye, the patient by fixing the ball with the seeing eye prevents any movement of the eye during the exposures and holds the visual line of the injured eye parallel with the plate. Two exposures are made upon one plate, metallic shutters protecting those portions of the plate which are not to be exposed to the rays.

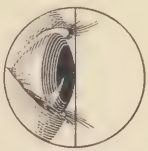


Fig. 383.—Image of cross-wire and cornea (W. M. Sweet).

The tube-holder contains the usual cylindric lead-glass shield for protecting the operator from the action of the rays, with the customary lead diaphragm. The central orifice of the diaphragm is covered with aluminum, which offers little obstruction to the rays, but lessens the risk of any unfavorable action of the rays upon the patient and guards against possible damage to the eyes in the event of breakage of the tube. The tube-holder slides upon a graduated rod, and the first exposure is made with the indicator at zero, in which position the rays pass in a direction corresponding with the horizontal plane of the eyeball. The second exposure is made with the tube at its farthest point to the right or left of the first position, depending upon which eye is to be examined.

Since the relative position of the tube in reference to the indicating

ball and the photographic plate remains fixed and known, the x-rays in passing through the eyeball follow a definite course, which is always the same for the two separate exposures. It is, therefore, possible to indicate on the localization chart the direction of the rays at the two exposures, and this has been done in the chart, a copy of which is reproduced in Fig. 384 reduced in size one-half. Only those lines representing rays 2 mm. apart are reproduced, but each line is drawn with the required amount of divergence to indicate the rays as coming from a point the distance of the tube from the photographic plate.

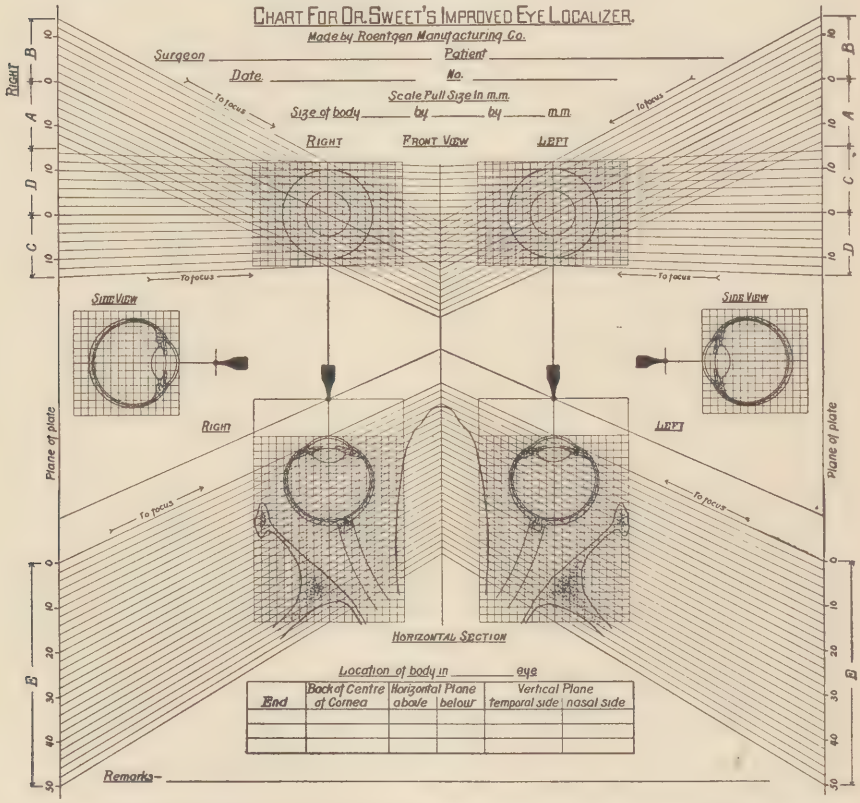


Fig. 384.—Localization chart, with lines representing course of the x-rays (one-half actual size) (W. M. Sweet).

In employing the new apparatus (Fig. 385) the jointed portion containing the upright tube, T, is raised, and the patient lies with the head on a platform of hard fiber, with a pillow beneath the shoulders and a small sand-bag under the head and neck. The upright supports for holding the head are now adjusted by means of the wheel 1, and the jointed part of the apparatus brought down in position. The indicating ball is now roughly adjusted until it is opposite the center of the cornea and about 12 or 15 mm. distant. The patient looks with the uninjured

eye into the mirror (*M*) and fixes upon the iris or cornea of the injured eye, or, better, upon the indicating ball in the center of the celluloid disk. The indicating ball is now adjusted directly over the corneal center by means of the wheels 2 and 3, and the correctness of the position verified by observation through an opening in the mirror (*M*). The operator then adjusts the light of the small electric lamp so that the side of the nose next the injured eye is illuminated, but the light is not thrown into the eye. With this area lighted it is possible, through the telescope (*T*), to note when the cross-wire is exactly tangent with the summit of the cornea. The movement necessary to secure this position of the wire is made by means of the adjusting wheel 4. When the image

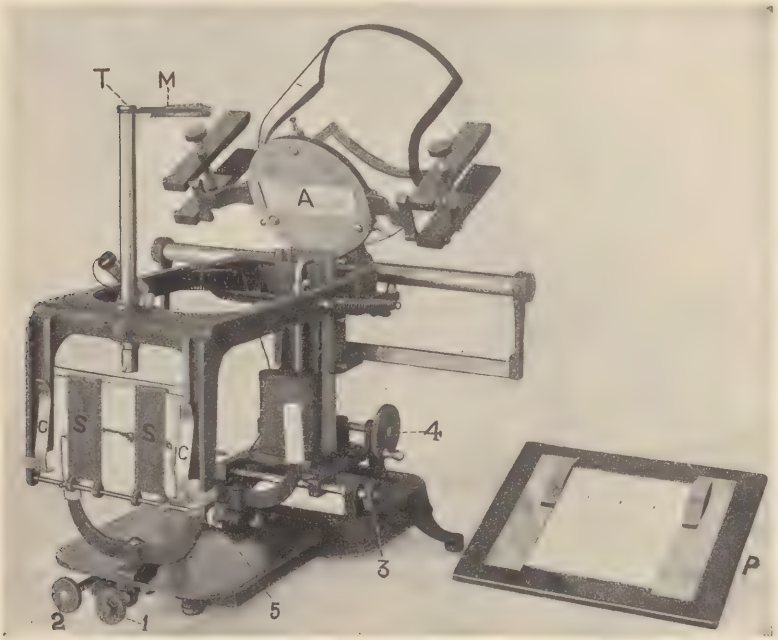


Fig. 385.—Sweet's apparatus for localizing foreign bodies in the eyeball.

of the cross-wire touches the image of the corneal summit, the indicating ball is exactly 10 mm. from the eyeball.

The photographic plate is inserted beneath the spring clips (*C*, *C*), the shutters (*S*, *S*) moved so that the center area is open, and the tube-holder adjusted to the zero point on the sliding scale. The current is turned on, and one exposure made. The tube-carriage is then moved to the limit of the sliding rod, always in the direction of the chin of the recumbent patient (to the end marked *R* if the radiographs are made of the right eye, and to *L* if of the left eye). The upper shutter is moved to cover the exposed central portion of the plate and uncover the upper unexposed portion. The current is again turned on and the second exposure made.

After the plate is developed it is placed in the frame *P* (Fig. 385), containing the key plate or focal coördinates (Fig. 386), with the film side of the radiograph next to the key plate. The radiograph is moved until the shadow of the indicating ball of the first exposure is in apposition with the middle ball on the key plate and the heavy horizontal line of the radiograph parallel with the horizontal line on the plate. Holding the frame to the light, there is noted the position occupied by the shadow of the foreign body with respect to the vertical lines of *C* and *D*. A reading is made of the line or lines which pass through the body, and this is transferred to the corresponding lines of the *C* or *D* scale of the chart, to the right or left side, depending on which eye is under examina-

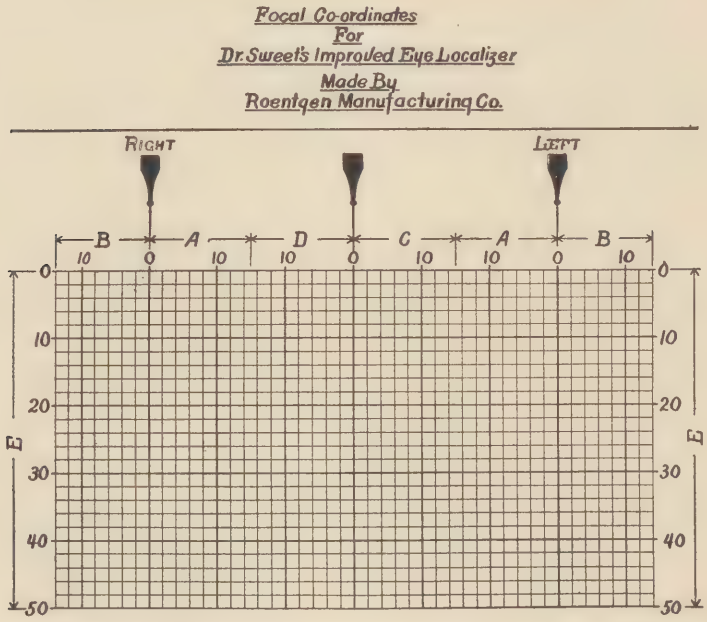


Fig. 386.—Plate showing focal coördinates (three-fourths actual size) (W. M. Sweet).

tion. Without moving the plate the *E* reading is similarly made and transferred to the chart. To take the *A* or *B* reading the plate is shifted slightly until the image of the indicating ball on the second exposure coincides with the "Right" or "Left" ball of the vertical coördinates *A* or *B*. The line or lines of the *A* or *B* coördinates which cross the shadow of the body are noted and indicated on the *A* or *B* lines of the chart. The horizontal coördinate *E* should be the same in both readings. If the focus point on the anode of the tube was accurately set by the cross-lines on the lead-glass shield of the tube-holder, the images of the indicating ball on the plate will coincide simultaneously with those on the transparent key plate, and it will then not be necessary to reset the plate to read the position of the *A* and *B* coördinates.

After the three readings have been transferred to the chart, the point of crossing of the *A* or *B* and the *C* or *D* lines is found, which gives the location of the foreign body in reference to the front view of the eyeball, indicating its situation above or below the center of the cornea and to the nasal or temporal side of the vertical plane. Where a vertical line from this point crosses the *E* reading on the horizontal section of the globe it gives the depth of the body in the eyeball or orbit. In bodies of large size both ends should be localized to give the position in which the body rests in the globe. The situation of the body

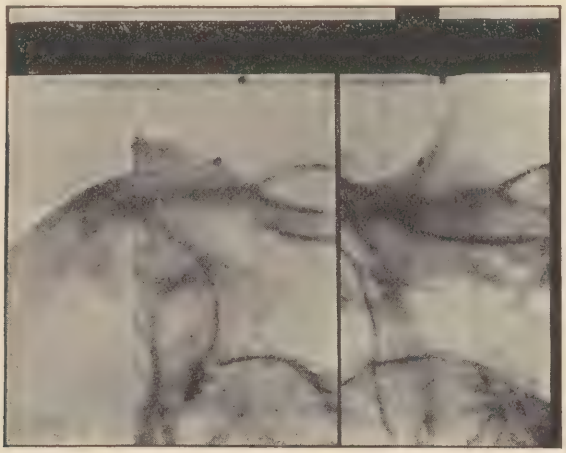


Fig. 387.—Radiograph of foreign body in eye (three-fourths actual size) (W. M. Sweet).

on the side view is determined by transferring its measured depth from the horizontal section and its distance above or below the horizontal plane from the front view localization.

The accuracy of the localization depends only upon the care with which the operator adjusts the indicating ball opposite the center of the cornea and at the definite and fixed distance from it. After the exposures are made and the plate developed, the determination of the situation of the foreign body is simply a question of reading from a key plate and transcribing these readings to the chart.

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